

THE BRITISH
JOURNAL OF SURGERY

THE BRITISH JOURNAL OF SURGERY

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GENERAL INDEX TO VOLUME XXII

SUPPLEMENT ATLAS OF PATHOLOGICAL ANATOMY

FASCICULUS A GANGRENE FIBROCYSTIC DISEASE OF BONE MISCELLANEOUS CONDITIONS

INTRODUCTION TO VOLUME XXII

THE BRITISH JOURNAL OF SURGERY has now completed its twenty-first year. The birth was attended by anxiety and even in some quarters by apprehension. Those who were responsible for its appearance were, however, deeply convinced that a journal representing the work of British surgeons would not only be a record of progress in this country and in our Dominions but would also prove to be an incentive and encouragement to all those engaged in pursuing the science or practising the art of surgery. It was decided that foreign contributions, having their own channels for publication, should not appear in our pages, which were to be strictly reserved for our own people in all parts of the world.

The infancy of our journal had to contend with difficulties inseparable from war time, but wise counsels prevailed, publication was continued, and many of the new experiences gained during the war first found full expression in our pages.

We have not rested content with pursuing our original designs. The reviewing of the best works on surgery published in any part of the world was from the outset a feature of each number published, in later years the less important works have received briefer notice so that we might concentrate upon those works regarded by us as being of greater value.

It was not long before the need was felt of adding to the value of each number by the publication of a supplement dealing with morbid anatomy, illustrated by specimens chosen from the more important museums of this country, and specially from the incomparable Hunterian Museum of the Royal College of Surgeons. Our desire indeed has been to create, in the course of years, an *ATLAS OF PATHOLOGICAL ANATOMY* whose contents should be representative of our great possessions, some of which were inherited from John Hunter himself and were the work of his own hands.

THE BRITISH JOURNAL OF SURGERY

A further ambition now awaits attainment. Those who have recently been engaged in the practice of surgery have realized that both the science and the art of human surgery are dependent, increasingly dependent, for their further advance upon research carried out in all possible directions, and not least upon animal experiment. Owing to the generosity of many friends, and notably of our wise benefactor Sir Buckton Browne, new and enlarged opportunities have been created for the extension of research work at the Royal College of Surgeons in new laboratories and at the Buckton Browne Research Farm erected in the grounds of Charles Darwin's house at Downe, in Kent. Young and competent research workers as well as senior men are there carrying out investigations destined to extend the range of surgery, to correct mistakes in old methods, to guide the surgeon in new activities, and to explain and illustrate many of the clinical experiences of surgeons, hitherto little understood. Our desire and firm intention is to encourage that close relation between clinical experience and experimental methods which is the sure foundation for the future advance and greater promise of our surgical work. We are inaugurating in the present issue a section dealing with this new work, and by so doing hope to bring home to all who read the JOURNAL the fact that we are concerned not merely with the details, however interesting or important, of our craft, but with a sincere effort to advance the science of the profession for whose welfare we exist.

Kroghian.

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IPSISSIMA VERBA

By SIR D'ARCY POWER, K B E, LONDON

II. JOHN HUNTER'S EXPERIMENT

EVERY surgeon knows that John Hunter based his teaching that syphilis and gonorrhœa were manifestations of a single virus and thereby threw back the scientific knowledge of both diseases for nearly a century. Few surgeons have read the account of the disastrous experiment on himself. It is contained in Part vi, Chapter 2, Section ii, p 324, of the *Treatise on the Venereal Disease* (London, 1786), and is in the following words "*of the Lues Venerea*" —

"To ascertain several facts relative to the venereal disease the following experiments were made. They were begun in May 1767

'Two punctures were made on the penis with a lancet dipped in venereal matter from a gonorrhœa, one puncture was on the glans, the other on the prepuce

"This was on a Friday, on the Sunday following, there was a teasing itching in those parts which lasted till the Tuesday following. In the meantime these parts being often examined, there seemed to be a greater redness and moisture than usual, which was imputed to the parts being rubbed. Upon the Tuesday morning the parts of the prepuce where the puncture had been made were redder, thickened and had formed a speck, by the Tuesday following the speck had increased and discharged some matter, and there seemed to be a little pouting of the lips of the urethra, also a sensation in it in making water, so that a discharge was expected from it, the speck was now touched with lunar caustic and afterwards dressed with calomel ointment. On Saturday morning the slough came off, and it was again touched, and another slough came off on the Monday following. The preceding night the glans had itched a good deal, and on Tuesday a white speck was observed where the puncture had been made, this speck when examined was found to be a pimple full of yellowish matter. This was now touched with the caustic, and dressed as the former. On the Wednesday the sore on the prepuce was yellow, and was therefore again touched with caustic. On the Friday both sloughs came off, and the sore on the prepuce looked red, and it's basis not so hard, but on the Saturday it did not look quite so well, and was touched again, and when that went off it was allowed to heal up, as also the other, which left a dent in the

* The Fridays in May, 1767, were the 1st, 8th, 15th, 22nd, and 29th
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glans This dent on the glans was filled up in some months, but for a considerable time it had a bluish cast

"Four months afterwards the chancre on the prepuce broke out again, and very stimulating applications were tried, but these seemed not to agree with it, and by letting it alone it healed up This it did several times afterwards, but always healed up of itself That on the glans never did break out, and herein also it differed from the other

"While the sores remained on the prepuce and glans, a swelling took place in one of the glands of the right groin I had for some time conceived an idea that the most effectual way to put back a bubo was to rub in mercury on that leg and thigh, which would send a current of mercury through the inflamed gland, this afforded a good opportunity of making the experiment I had often succeeded in this way, but now wanted to put it more critically to the test The sores upon the penis were healed before the reduction of the bubo was attempted A few days after beginning the mercury in this method the gland subsided considerably It was then left off for the intention was not to cure it completely at present The gland some time after began to swell again, and as much mercury was rubbed in as appeared to be sufficient for the entire reduction of the gland, but it was meant to do no more than to cure the gland locally, without giving enough to prevent the constitution from being contaminated

"About two months after the last attack of the bubo, a little sharp pricking pain was felt in one of the tonsils in swallowing anything, and on inspection a small ulcer was found, which was allowed to go on till the nature of it was ascertained and then recourse was had to mercury The mercury was thrown in by the same leg and thigh as before, to secure the gland more effectually, although that was not now probably necessary

"As soon as the ulcer was skinned over the mercury was left off, it not being intended to destroy the poison, but to observe what parts it would next affect About three months after, copper-coloured blotches broke out on the skin, and the former ulcer returned in the tonsil Mercury was now applied the second time for those effects of the poison from the constitution, but still only with a view to palliate

"It was left off a second time, and the attention was given to mark where it would break out next, but it returned again in the same parts It not appearing that any further knowledge was to be procured by only palliating the disease a fourth time in the tonsil, and a third time in the skin, mercury was now taken in a sufficient quantity, and for a proper time to complete the cure

"The time the experiments took up, from the first insertion to the complete cure, was about three years

"The above case is only uncommon in the mode of contracting the disease, It proves that matter from a gonorrhœa will produce chancres

"It makes it probable that the glans does not admit the venereal irritation so quickly as the prepuce The chancre on the prepuce inflamed and suppurated in somewhat more than three days, and that on the glans in about ten "

* The practice in 1767 was to apply a mercurial plaister on the part, or to rub in mercurial ointment on the part, which could hardly act by any other power than sympathy [Hunter's footnote]

Although the experiment was begun in 1767 it was not until 1786 that Hunter published this account of it in the *Treatise on the Venereal Disease*. It is remarkable that so acute an observer should have considered himself cured and did not connect his subsequent ill health with a persistence of the disease. The term 'Hunterian chancre' or 'hard sore' soon came into general use in England, though his views were not accepted universally. Richard Carmichael as early as 1814 was teaching that there were different kinds of venereal disease, that they were produced by different poisons, and that they did not all require mercury for their cure. Philippe Ricord repeated Hunter's experiment in 1836 and showed that gonorrhœa was distinct from syphilis. Bassereau in 1852 and Clerc in 1854 taught that soft sores were not due to the same poison as that which produced gonorrhœa or syphilis. It was not until the birth of bacteriology that the age-long controversy was at last ended. In 1879 Albert Neisser, of Breslau, recognized the gonococcus, in 1889 Ducrey announced the discovery of a causative bacillus in pus taken from a bubo associated with soft sores, and on March 3, 1905, Fritz Schaudinn pointed out to Paul Erich Hoffmann the *Spirochæta pallida* as the infecting organism of syphilis.

THE EXAMINATION OF FRESH TISSUES BY THE WET-FILM METHOD

By LEONARD S DUDGEON, C M G, C B E

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THE use of wet films as a method of pathological investigation was introduced by Dudgeon and Patrick¹ in a paper published by them in the BRITISH JOURNAL OF SURGERY in 1927. A method of rapid diagnosis of new growths had previously been perfected by E H Shaw,² and in his hands the results had been successful. The wet-film method was tried in a series of 200 consecutive cases of new growths and inflammatory tissues, and it was found that not only was it possible to make a complete and accurate diagnosis on the films, but that the specimens obtained were so perfect that the details of cell structure could be appreciated in a way not possible in the ordinary section. Encouraged by these results the work has been continued since that time, and this paper represents an analysis of over 1000 cases.

Dudgeon and Patrick emphasized the value of wet films as follows: "we consider the advantages are the beautiful preparation of individual cells and fragments of tissue which are seen in the films, the simplicity of the technique, and the small amount of material required for the preparation of the films." The additional experience gained since that time has confirmed these views, and it is suggested that the practical value of the method is much greater than is generally realized. It may be argued that the existing methods are sufficient for the purpose, but one cannot fail to be impressed by the possibilities which present themselves and which are not open, for one reason or another, to these methods.

The fixing of the wet cells with Schaudinn's fluid is very satisfactory and they are so little damaged by the process that they are presented as nearly as possible in their true form.

A very wide area of the original tissue can be covered. This point is of importance, for not only does it enable the pathologist to study normal and abnormal cells in the same preparation, but in those conditions where differences of histological structure occur in various parts of a specimen, they are more likely to be represented in a wet film than in a section taken from a small area.

It must be emphasized that the method has been found to be applicable to all types of lesion, and although it was originally devised as a way of obtaining a rapid diagnosis in the case of tumours it has since been used for the more detailed study of every tissue which could be obtained in a fresh state.

The study of tissues as a whole has been shown to be of great interest, and in the later part of this investigation the routine of making wet films from tissues far removed from the main lesion was adopted. In the breast, for instance, where radical amputation had been done for carcinoma, wet films were made from the

WET-FILM EXAMINATION OF TISSUES

5

muscle, the skin, and outlying parts of the gland, and it was found that in a large percentage of cases malignant cells were discovered lying amongst the normal tissues. Table I gives the actual results of this investigation.

Table I

ORIGIN OF TISSUE	CASES EXAMINED	CARCINOMA CELLS FOUND IN APPARENTLY NORMAL TISSUES
Breast	37	26
Stomach	8	3
Rectum and colon	9	6
Uterus	3	3
Tongue	1	1
Total	58	39

It will be seen that the total number of cases investigated was 58, and in 39 (67.2 per cent) carcinoma was found in apparently normal tissues which had not reacted to its presence. The malignant cells were often isolated from each other or in small clusters, but they differed so markedly in their appearance and behaviour from normal cells that the diagnosis could be made with certainty. By this method, therefore, not only can the spread of disease be investigated, but a more accurate judgement as to the extent of a lesion can be formed.

In the operating-theatre most surgeons form their opinions on the macroscopic appearances of the conditions they meet, but in any case of doubt, where the immediate treatment depends upon an exact diagnosis, it is well to remember that this can be given in ten minutes, and it is as exact in its rendering as the more prolonged investigation by means of sections. As an example of the value of a wet film to the surgeon the case quoted by Wrigley⁷ is interesting. The conclusion had been reached that a certain abdominal tumour was malignant, but a wet film was taken and the diagnosis of cystadenoma of the ovary given immediately. The operation was therefore continued and the tumour was successfully removed.

The materials necessary for making wet films can be taken from place to place in a very small box, and the method has been used successfully on many occasions away from the laboratory. In the out-patient department and wards of a hospital or a consulting-room it can be applied particularly to the diagnosis of doubtful lesions of the skin and exposed parts of the body, and consequently loss of time may be avoided, a diagnosis can be given immediately, and appropriate treatment can be instituted.

It remains to be said that with these advantages there are certain limitations. There is no doubt that a sound knowledge of pathological processes, as they are seen in ordinary sections, is required, and to this must be added a great deal of practice in studying wet films made not only from diseased areas, but from the normal tissues in the vicinity. Again, the practical difficulty of obtaining the

material in a completely fresh condition is very real, and the specimens must not be put into water, saline, or preservatives

TECHNIQUE

The method which has been used is practically the same as that described in the original paper¹ It has been emphasized that the correct interpretation of a wet film depends on the perfect presentation of the individual cells, and this can best be obtained by observing the following technique, the minor points of which are discussed in detail The actual scraping of the tissue needs careful handling Absolutely fresh material is essential Water, saline, preservative, or fixing solution makes the tissue useless for wet films The same is true of post-mortem material, for in this the earliest changes of autolysis are sufficient to distort the normal appearances and make diagnosis difficult or impossible

Take the specimen, therefore, as soon as it has been removed by the surgeon and examine it carefully with the naked eye to determine the part most affected by the disease This should present but little difficulty, and yet it is surprising how often a film can be made from a normal area in the vicinity of the lesion or from one in which autolysis or degeneration is so far advanced that the cells cannot be recognized As an illustration of this practical point the following case is instructive A patient attended the Out-patient Department complaining of a chronic ulcer on the dorsum of the hand, as the diagnosis was in doubt a wet film was prepared from the surface of the lesion, and on examination this showed acute inflammation and masses of degenerating surface epithelium A second film made from a small piece of tissue removed under local anæsthesia from the edge of the ulcer revealed a squamous-cell carcinoma Particular care is necessary in the case of films made from lymphatic glands

It is well to dry the tissue with a piece of gauze, for the red blood-cells which are certain to be present in large numbers are not needed in the preparation, and the tissue fluids which exude from the cut surface upset the evenness of the film by lying over the individual cells and producing unequal staining

The film is made by scraping the tissue with a sharp knife so that the cells are collected upon the blade and can be transferred immediately to a glass slide A smear is then made in the same way as a blood-film

The optimum thickness is a matter of experience, but on the whole films of very cellular structures, such as lymphatic glands or malignant growths, should be thin, whereas in the case of chronic mastitis, for instance, where the acini are few and far between, a wide area must be covered and a fairly thick film prepared Once the film has been made, no time must be wasted in removing from the slide those parts which are too thick or lumpy, for it is essential that it be placed in Schaudinn's fluid immediately, before drying occurs

This fluid is prepared by adding one volume of absolute alcohol to two volumes of a saturated aqueous solution of mercuric chloride The mixture can be kept indefinitely as a stock solution, but immediately before use glacial acetic acid to the strength of 3 per cent must be added⁴ If the acid is added to the stock solution a long time before it is used for fixing the wet films, it will be found that there is a tendency for crystallization to occur The best fixation is achieved by leaving the slide immersed in this fluid for as long as twenty minutes,

when, however, a rapid diagnosis is needed a minimum of two minutes is all that is necessary. The slide is then transferred to methylated spirit to which has been added a few drops of tincture of iodine. The iodine has the property of removing the excess of mercuric chloride by converting it into the more soluble mercuric iodide. A washing in distilled water prepares the slide for staining.

It has been found that Mayer's hæmalum gives the most constant results as a nuclear stain. It is made by dissolving 1 gram of hæmatoxylin in 1000 c.c. of distilled water. The solution must be heated to get the hæmatoxylin to dissolve properly. To this are added 0.2 gram of sodium iodate, 50 gram of ammonia alum, and finally 20 c.c. of glacial acetic acid. The advantages of this particular stain are that it is much cheaper than the hæmalum which is made from hæmatein, and the sodium iodate enables it to be used immediately without having to wait for 'ripening' to take place. The slide is left in the stain for about two minutes or less, depending upon the strength of the solution. It is then transferred to tap water. In the water the colour changes from a reddish purple to blue, and the slide must be left until 'blueing' has taken place. During this time the thick or uneven parts of the film can be removed from the slide by touching them with a needle. As a counterstain a weak solution of eosin is used.

The slide is then taken through the alcohols to xylol in the usual way and mounted with Canada balsam and a cover slip.

Several stains besides hæmalum and eosin have been tried, but we have not found any which bring out the important features in a way superior to these, and only in special cases has it been necessary to use other methods.

POINTS IN THE INTERPRETATION OF WET FILMS

Some account of the various pictures presented by the more common pathological conditions will now be given.

Normal epithelium presents a most perfect picture. The cells are seen either in flat plaques like the stones of a finely tessellated floor, as individual cells, or as groups lying side to side. They are easily recognized since the staining reaction and general outlay are remarkably constant. The only exception to this are the cells found in the superficial strata of desquamating squamous epithelium, and these are often irregular in shape and separate from each other. On close examination of normal epithelium the following points can be observed: the similarity in size and shape of the cells, the fact that the nuclei are placed in like positions with regard to the remainder of the structure, the very fine reticular arrangement of the nuclear chromatin, giving the appearance of even consistency to each nucleus, and finally the tiny pink nucleoli. Further, it will be seen that where several cells are joined together in a plaque the cytoplasm stains differently towards the margin of each cell, for whereas in the main body of the cell it is blue or purple in colour, towards the edge there is a lighter area, which is itself bounded by a fine dark line. It is this dark line, lying as a sort of fence between adjacent cells, which is responsible for the tessellated appearance described above. Isolated cells appear to be limited by a definite cell membrane.

In the plaques the outline of the cells is angular and they fit against one another like the chambers of a honeycomb, and the edges may show nuclei and protoplasm extruded leaving delicate fringes of the ruptured cell membranes.

It is interesting to note that Shattock and Dudgeon,³ in a paper read before the Pathological Section of the Royal Society of Medicine in 1914, included a diagram which was described as follows "A plaque of carcinomatous cells detached from one of the intact eminences of a tumour of the colon by lightly rubbing a cover-glass over it, examined in glycerine" The method, it will be noted, is not that which is described in this paper. The diagram actually shows a plaque of perfectly normal epithelium and demonstrates the characteristic features mentioned above. It shows incidentally how easily a piece of tissue can be taken from the wrong place. In this case it was assumed that the cells must be malignant in view of the site from which they were thought to have come.

The stroma of a tissue is represented, but the cells are not easy to differentiate, and except in the case of some malignant growths they are of limited interest. Arteries and veins cannot be distinguished as such, but capillaries are sometimes beautifully shown, and these have been observed to perfection in many tissues, and were specially noticed in gliomata and lesions of the central nervous system.

To this picture of normal cells the features of inflammation may be added. The polymorphs of acute inflammation are particularly easy to identify, for they are large and natural as in a blood-film. The same applies to the large mononuclear cells, plasma cells, and lymphocytes of chronic inflammation.

The phagocytic properties of polymorphs, large mononuclear cells, and malignant cells of all types have been repeatedly observed—special reference to the malignant cells will be made in another section of this paper—and the various types of giant cells which are typical of special conditions can be quickly recognized.

As an example of the application of these points a wet film made from a small tumour in the subcutaneous tissues of a patient's leg may be quoted. It showed a large number of foam cells, many of which were packed with pigment, masses of small round cells, and giant cells of the foreign-body type. A second film was made from the tumour and stained to show free iron, and this being positive a diagnosis of organizing hæmatoma was correctly given. It may be remarked that in cases of doubt as to the nature of a pigment, melanin can be quickly distinguished from blood pigment, since it does not give the free iron reaction.

Normal epithelium can be modified considerably by the presence of acute inflammation. In this condition the cells may be large and more swollen than normal, the boundaries less distinct, and the cells themselves separate from one another. In addition there may be variations in the size and shape of the nuclei. Some of these features are due to degeneration, and this is shown by pink staining of the cytoplasm. Close inspection, however, reveals the fine reticular arrangement of the nuclear chromatin, and remnants of the tessellated plaques will nearly always be found. In these cases it is particularly the degenerating individual cells which are apt to be misleading.

The features of tuberculosis are readily defined. In support of this it can be said that the diagnosis has been made from the wet film when this was not possible from the corresponding section, and the accuracy of the verdict has been upheld by the subsequent clinical progress of the patient.

Lymphadenoma is easy to diagnose in wet films, and the 'rapid technique' is particularly useful in cases where the diagnosis has to be made from tuberculous adenitis, for the surgical treatment of the glands is different in the two conditions.

There is little to say as regards the distinctive features of benign neoplasms except that in these tumours all the characters of normal cells of the same tissue are shown and that there are usually more cells in the field. A polyp of the rectum, for instance, shows more epithelial cells and less stroma than a piece of rectal wall. Since, however, this distinction is only relative, it cannot be held to be characteristic of benign tumours.

In the diagnosis of wet films made from tumours the main point at issue is the simple or malignant nature of the cells. Now it has always been strongly held that "in the appearance of a cell from a cancer there is nothing characteristic of the disease, nothing that would lead a pathologist to identify it as a malignant cell. Cancer can only be identified in sections showing the relation of cells to each other in a group." This is the view expressed by Bland-Sutton⁵ in 1922, and it is evident that it still holds the field, for in 1932 Ludford⁶ writes "an extensive search has been made for any difference between normal and malignant cells which might help to explain the cause of malignant growth, or reveal some weak spot in the armour of the cancer cell towards which therapeutic attack might be directed. It has been pointed out that we have no definite morphological criterion of malignancy, but certain differences between normal and malignant cells have been established." He goes on to say that these differences are concerned with the reaction of malignant cells towards acid vital dyes.

From the study of wet films, it seems certain that malignant cells are different in appearance from normal cells and that their special histological features are so striking that they can be picked out as isolated units in a large field. The truth of this statement is borne out by the following figures from the series under consideration.

Total number of wet films of malignant disease examined	469
Total number diagnosed as malignant	462

It is not suggested that a solitary cell could be pronounced malignant on its own merits, but in a field where benign cells are present for comparison the diagnosis is not difficult. It depends upon the special characters of the individual cells and not upon the presence or absence of invasion of normal tissue, although if this point is shown in the wet film it is taken as corroborative evidence. The features which in a wet film suggest that a cell or group of cells is malignant can be defined as follows —

Malignant cells stain more deeply than their benign prototype. This is due to the fact that the nuclei have a greater affinity for hæmalum. They vary in size and shape, they are larger than normal, and their position relative to the surrounding cytoplasm is in no way constant. The nuclear chromatin is arranged in thick, irregular, and deeply staining bundles, and the fine reticular arrangement of normal cells is rarely seen. The nucleoli are much larger than normal, they are sometimes multiple, and stand out as purple or pink dots in the sphere of the nucleus.

Mitosis is beautifully demonstrated, but it cannot be regarded as characteristic of malignant cells alone. It has been observed in many benign conditions, and all that can be said is that it is most frequently found in malignant cells. It is worth noting, however, that in tumours such as papillomata of the urinary system, which frequently prove to be malignant, mitosis is very much in evidence. Large

numbers of mitotic figures are presumably an indication of the rapid proliferation of the cells concerned, but the cases of carcinoma of the breast which were followed up in detail and which are reported in this paper showed quite definitely that the picture was not necessarily of bad prognostic significance

The actual size of the cells varies enormously, so that extremely large and very small ones can be found lying side by side in the same preparation. The larger cells are often multinuclear.

Malignant epithelial cells do not stick together in regular formation, and placards are the exception rather than the rule. In those cases where placards are found the cell boundaries are usually not apparent and the appearance is that of a syncytium from the edges of which nuclei, devoid of cytoplasm, can be seen breaking away. In some films, however, the cell boundaries are prominent, and in spite of this the cells separate from each other. This was particularly noticed in a film made from a carcinoma of the stomach, in which the boundaries were very obvious and yet the majority of the cells were isolated from each other, several placards were present, but these were different from those of benign epithelium, for the regular geometrical arrangement was lacking.

It is a feature of benign epithelium that the sheets of cells are not usually more than one layer in thickness, and in films of malignant tissue the appearance of placards is often due to the cells lying on top of each other in heaps. In normal epithelium the cells stick together because there is some cement substance between the individual units. This substance is capable of holding them together by itself, for in wet films made from normal epithelium many cells are presented in rows and it is easy to see that there is no basement membrane supporting them. Now there is evidence that the cement substance is either not represented in malignant tissue or differs in nature from that of benign epithelium, for malignant cells separate very completely from each other and many are disrupted so that the nuclei are entirely devoid of surrounding cytoplasm.

In malignant growths there are numbers of degenerating cells. In the early stages they are swollen and the outlines are distorted by alterations in the structure of the cytoplasm, which stains more pink as the condition advances. The nuclei, which at first are very large, gradually shrink and the chromatin condenses into a solid mass which ultimately breaks up and disperses in many basophil granules. In some cases the nuclear membrane is destroyed before the cell disintegrates and small discrete blocks of chromatin can be seen lying at random in the cytoplasm. Inclusion bodies are found in all types of malignant cell, but are most prominent in epithelial tumours.

It has been pointed out by Carleton⁸ that "it is usual to regard phagocytosis as the perquisite of various motile cells which are generally regarded as mesodermal in origin", but that his conclusions lead him to suppose that "certain mammalian epithelia are capable on appropriate stimulation of responding by phagocytosis to the call made upon them". Phagocytosis is therefore not commonly seen in benign epithelium except under special conditions, and never in the same rampant form as in malignant epithelial cells. The special powers of phagocytosis possessed by malignant cells have been mentioned already. It is often surprising how much material can be taken up by a single cell, for it is not unusual to find many polymorphs in the cytoplasm. Phagocytosis appears to be most in evidence in those tumours which have been submitted to a course of radium or X-ray

treatment, and in the tumour cells polymorphs, red blood-corpuscles undergoing hæmolysis, tissue cells, pigment, and debris can all be seen

With these points in view it is often possible to give an opinion not only as to the malignant nature of a group of epithelial cells but also on the type of epithelium from which they originated. Great variation exists, however, and sometimes the finer points such as the difference between a columnar- and a spheroidal-cell carcinoma cannot be determined. The absence of stroma in wet films of carcinoma may lead to difficulty in distinguishing the condition from sarcoma, especially as the amount of cytoplasm in the cells cannot be taken as a guide one way or the other. Sarcoma cells show all the abnormalities of nuclear structure which are found in carcinoma but often to an exaggerated degree. The separation of the cells from each other is more marked, and there is seldom any clue in the wet film as to the origin of the tissue. In pigmented tumours the blood pigment must be distinguished from melanin.

There remain for consideration the rodent ulcers which are locally malignant, and here the characteristics of the film histology are intermediate between those of a carcinoma and a simple tumour.

SPECIAL SYSTEMS

In the following account the special points which have arisen during the course of this investigation are set out and discussed in detail under the appropriate organs or systems whence the specimens were obtained.

Alimentary System—The value of wet films has been particularly evident in the diagnosis of the various pathological conditions of the lips, mouth, and tonsils. A small piece of tissue can easily be removed under a local anæsthetic, and the diagnosis can often be determined at once. This has been done with success in many cases, and of these the following are quoted as examples. A patient came to the Hospital complaining of a large, painless, and punched-out ulcer on the dorsum of the tongue. The ulcer was judged to be a gumma which had broken down, or a carcinoma. The Wassermann reaction was found to be negative. A wet film had been made, however, and this showed the most perfect endothelial-cell proliferation, caseation, and giant-cell formation, so that a diagnosis of tuberculosis was given. Subsequently the patient was found to have pulmonary tuberculosis, and under appropriate treatment the ulcer healed. Again, a man came to the Out-patient Department complaining of a swelling of the right tonsil. The tonsil, on examination, was slightly bigger than normal, but it was quite soft on palpation and the diagnosis of malignancy was not considered at the time. A wet film was taken. The tumour proved to be an epithelioma and the patient was admitted shortly afterwards. The importance of this case is that in the ordinary course of events the diagnosis would have been in doubt until after admission, probably a month later, and this valuable time would have been wasted.

In wet films from the stomach some of the most interesting features have been found (*Figs 1, 2A*). There is probably no site in the body, except perhaps the endometrium, the gall-bladder, and the prostate, where the tessellated appearance of benign epithelium is presented so beautifully.

The cases of ulcer and carcinoma of the stomach which came for diagnosis were correctly reported upon in every instance. An interesting observation has

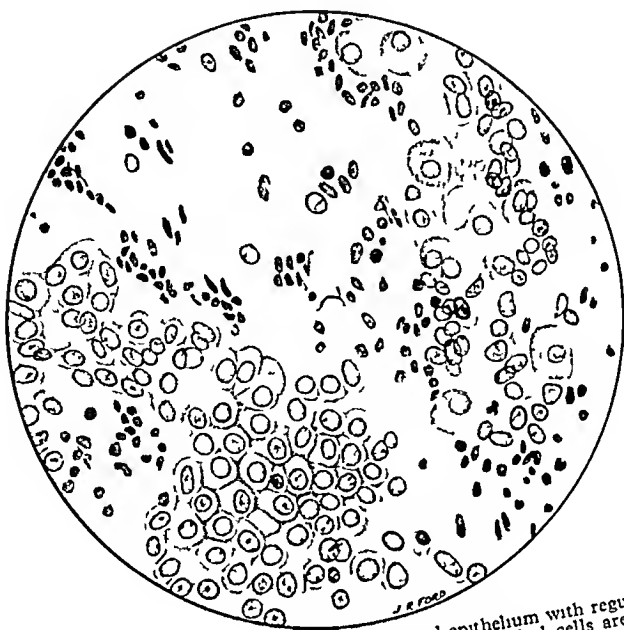


FIG 1—Gastric mucosa. Showing placards of normal epithelium with regular cell boundaries on the left of the picture on the right are the large syncytic cells. Red cells are scattered throughout the field ($\times 300$)

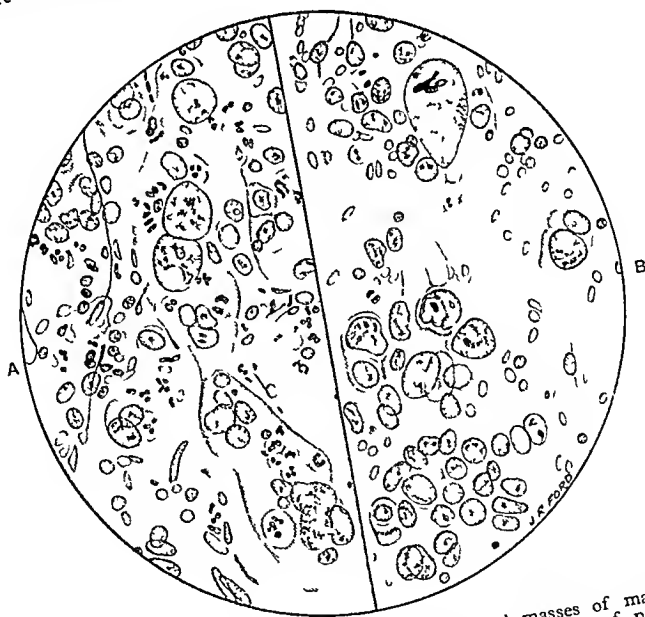


FIG 2—A, Stomach Carcinoma. Note the large syncytial masses of malignant cells. Some of the nuclei are very large and a considerable degree of phagocytosis of polymorphs is shown. B, Bladder Carcinoma. This diagram shows the variation in the shape and size of the cells, many of which have a large nucleus and very little cytoplasm ($\times 300$). (These two figures should be compared with Figs 1 and 3)

been made with regard to the oxyntic cells which have been supposed to secrete the hydrochloric acid of the gastric juice. These cells are often well seen in a wet film, they are usually arranged in small groups of five or six, and since they are larger than columnar epithelial cells it is easy to pick them out (*Fig 1*). In addition the nuclei may stain more pink than those of epithelial cells and the cytoplasm contains very fine eosin granules. In this series there were only 9 cases in which a test-meal had been done before operation, and in these particular cases the presence or absence of oxyntic cells was recorded as shown in *Table II*.

Table II

DIAGNOSIS	TEST-MEAL	OXYNTIC CELLS
Sarcoma	Total HCl 0.190	None
Carcinoma	No HCl Lactic acid +	None
Carcinoma	No HCl Lactic acid +	Oxyntics +
Carcinoma	No HCl Lactic acid +	Oxyntics + +
Gastric ulcer	Total HCl 0.113	Oxyntics + +
Carcinoma	No HCl Lactic acid +	None
Gastric ulcer	Total HCl 0.059	None
Gastric ulcer	Total HCl 0.33	Oxyntics + +
Carcinoma	No HCl Lactic acid +	Oxyntics + +

In addition to these there were 11 cases of gastric ulcer, in 10 of which oxyntics were found, and 2 of carcinoma, both showing the same thing. Oxyntic cells were present in films made from the stomach in cases of gastric carcinoma which have no free HCl in the gastric juice.

Urinary System (*Figs 2B, 3*)—Papillomata of the bladder and renal pelvis, which often prove to be malignant, may be diagnosed as carcinoma earlier in a wet film than in a section, and there were two occasions in the series on which this was possible. The diagnosis was subsequently confirmed by the clinical progress of the patients.

The report on a case of hypernephroma gives a good idea of the characteristic appearances of these tumours, wet films were made from two different areas. The 'red' area showed placards of large cells with small nuclei and clear cytoplasm together with numerous isolated cells of the same type, in addition there were large numbers of hæmolyzing red cells. The 'yellow' area was quite different and

* With higher magnification than has been used for *Fig 1* the eosinophil granules of the oxyntic cells can be clearly seen.

presented a solid mass of finely granular foam tissue, throughout which were numbers of solitary nuclei. The outline of the individual cells was indefinite, and the nuclei varied in size and shape.

The secondary deposits from malignant growths of the kidney are often very true to type, so that it is frequently possible to state from a wet film that the malignant epithelium is of the transitional-cell variety. In the following case the surgeon had operated upon a female with a large tender tumour in the upper part of the abdomen on the left side. On inspection of the abdominal viscera a malignant mass was found lying between the stomach, spleen, and kidney, in addition there were two collections of hæmorrhagic exudate, the one in the lesser sac and the other in the transverse mesocolon. It was impossible to state the exact site of the primary tumour, but the posterior wall of the stomach seemed to be the most

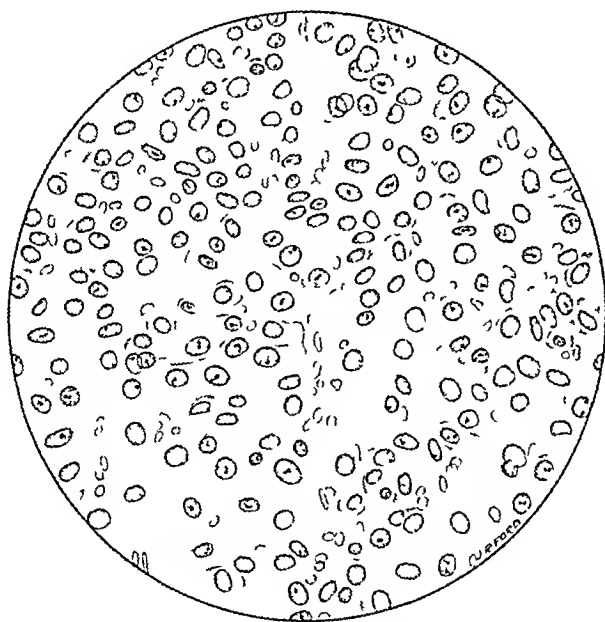


FIG 3.—Bladder Papilloma. This diagram shows the regular size of the individual cells, placards of cells, and the abundance of isolated cells, all with a large amount of cytoplasm. ($\times 300$)

likely place. A small secondary lying in the great omentum was removed for microscopy. Wet films made from this secondary deposit showed a transitional-cell carcinoma, and at the autopsy on the following day the kidney was found to be replaced by a mass of new growth which had started in the pelvis of the kidney and spread into the stomach wall and the spleen.

Benign enlargements of the prostate are very easy to diagnose, for the arrangement of the epithelial cells into placards is clearly shown, the tessellation is obvious even with a low power of the microscope, and the corpora amylacea are usually plentiful. In the original paper a drawing made from a wet film of an adenoma of the prostate was included. The features are so constant that malignant conditions can be quickly identified from benign. The similarity between sections made from adenoma of the prostate and certain conditions of the breast is well known,

in wet films the two varieties of epithelium are seen to be quite different in appearance, for that of the breast never demonstrates the tessellation to the same degree of perfection as the placards from the prostate

From the surgical point of view the danger of removing a prostate in the presence of cystitis is recognized, but it has been emphasized in an investigation of the 23 cases in this series in which the history of the progress after operation was available. There were 12 who did not show any evidence of inflammation in the wet film, and 10 of these patients had a normal convalescence, the other 2 died, the one of uræmia and the other of cardiac failure, but in neither was there gross infection in the bladder or kidneys. In the other 11 cases varying degrees of acute inflammation were recorded from the wet film. Two of the patients died from suppuration in the abdominal wall, and the remainder all developed septic complications of varying kinds.

The presence of acute infection in these cases was not sufficiently evident in the corresponding sections for comment to be made upon it.

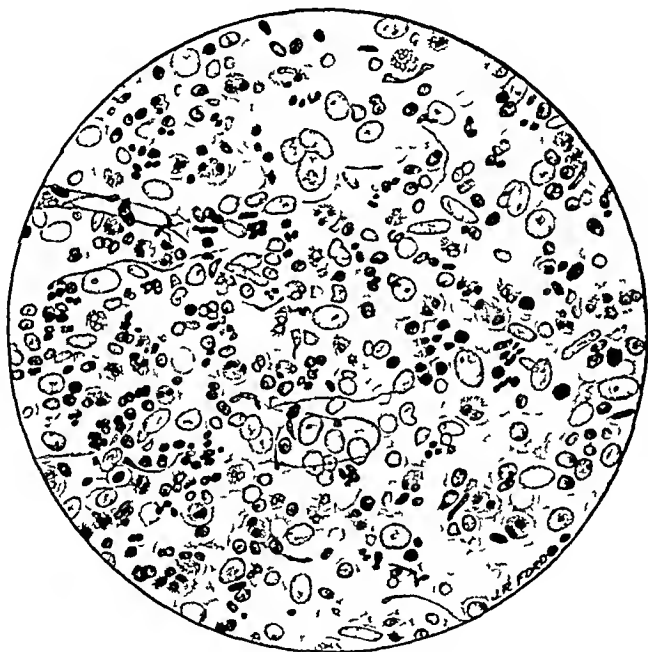


FIG 4—Lymph-gland Lymphadenoma Showing a considerable eosinophilia ($\times 300$)

Lymphatic System—A normal lymph-gland shows a highly cellular field of separate lymphocytes and a smaller number of large mononuclear cells. The nuclei of the lymphocytes, which are remarkably constant in size, shape, and staining reaction, take up the hæmalum well, and the cytoplasm is not shown. The stroma is not represented. It is sometimes difficult in a case of tuberculous lymphadenitis to pick out the best area of gland from which to make a wet film, for one is apt to get too much caseous material and debris by scraping the wall of an abscess cavity, and a film made from a neighbouring gland may show nothing more than chronic lymphadenitis. The best practice, therefore, is to scrape

suspicious patches of the gland and make a film which includes cells from a number of different places

The picture of lymphadenoma is one of the most satisfactory shown by this method, for in almost every instance the wet film is very much better than the corresponding section. The picture is one of a lymph-gland in which there is evidence of endothelial-cell proliferation, to this are added the two varieties of lymphadenoid giant cell and a number of eosinophils (*Fig 4*). The cells are all separate and consequently they can be studied individually. In every case of lymphadenoma there have been some eosinophils to be found, and it is worth noting that on the two occasions when a faulty diagnosis was made the wet film presented some of the characters of lymphadenoma but there were no eosinophils. The differential diagnosis rests between chronic lymphadenitis, tuberculosis, and endothelioma of the lymph-gland.

The diagnosis of chronic lymphadenitis is often one of exclusion. The presence of many cells in addition to the lymphocytes suggests some pathological process, but the absence of sufficient evidence pointing towards tuberculosis or other specific condition often necessitates a verdict of chronic lymphadenitis, and it can safely be said that if the smear has been properly made, this diagnosis will be as complete as the diagnosis from the section. The subsequent history of the cases in this series in which chronic lymphadenitis has been diagnosed has been followed up, and, as far as one can tell, these patients have not ultimately developed tuberculosis, lymphadenoma, or malignant disease, so that the true cause of the lesion remains obscure.

Muscle (*Fig 5*), Connective Tissues, and Bone.—The majority of lesions in this section have been of a neoplastic nature, but several organizing hæmatomata of muscle and fat have been correctly diagnosed. The only points which arise in connection with sarcomata are that the actual type of cell is often reported upon differently in the wet film and in the section—that is, a tumour in the film may show the features of a round-cell sarcoma, whereas the section may be returned as a fibrosarcoma.

Central Nervous System—Specimens from the central nervous system have not been sufficiently numerous for definite conclusions to be formed, but the fixation of the films in Schaudinn's fluid appears to give valuable information in differentiating the 'gliomata'. An investigation of these tumours was started in conjunction with the late Sir Percy Sargent, but the work has had to be abandoned. Hæmalum and eosin do not bring out the finer points which are claimed for the more complicated methods, though the detail in a wet film is very much greater than that of an ordinary section. A spongioblastoma multiforme was reported upon thus: "A malignant growth with cells of the sarcoma type. Very little cytoplasm around the nuclei. Capillaries beautifully shown. Giant cells and foam cells present. Delicate fibrils can be seen running in all directions from the malignant cells."

Breast (*Fig 6*)—The most important group of wet films which have been made are from the breast. The total number of cases examined was 212, and *Table III* (p 18) gives the figures in detail.

Normal breast tissue shows a great deal of fat and very little epithelium, in fact, except in the vicinity of the nipple it is quite possible to miss the epithelial elements altogether in a scraping. In lactation, however, large epithelial cells are abundant and these contain the characteristic globules.

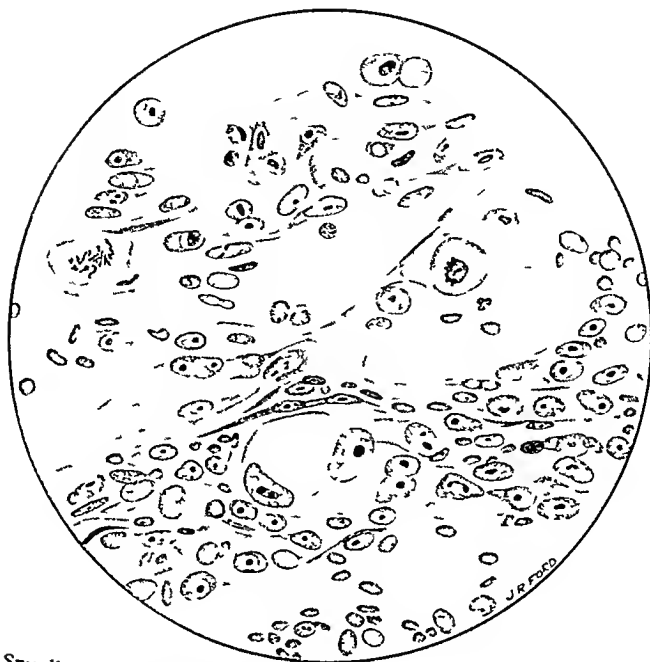


FIG 5—Muscle Spindle-cell sarcoma Note the variation in the size of the cells and the large amount of cytoplasm ($\times 300$)

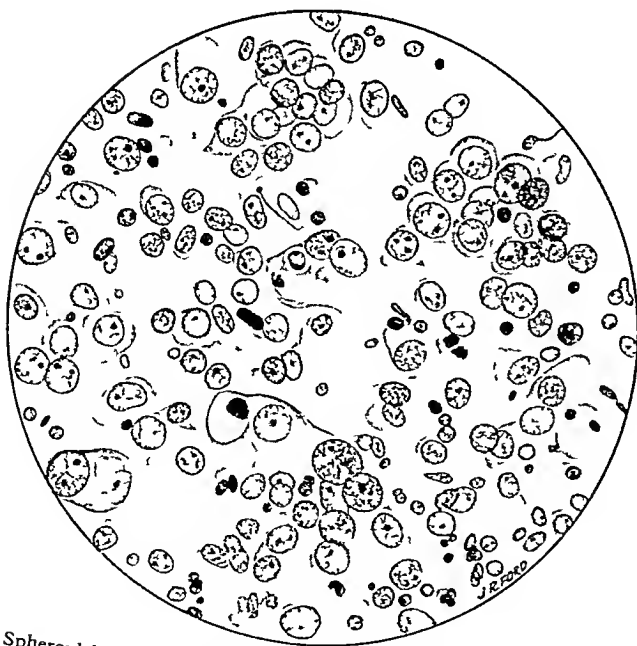


FIG 6—Breast Spheroidal-cell carcinoma showing syncytial masses and large amount of cytoplasm in the cells ($\times 300$)

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It is sometimes difficult to differentiate a case of chronic mastitis from a normal breast in a wet film, but in the former, although the epithelium is scarce, there is a fibrous-tissue stroma, there are groups of partly degenerate cells from the cyst walls or dilated ducts which stain pink with the eosin and are much larger than the normal epithelium, and a number of large foam cells. A diagnosis of 'simple' as opposed to 'malignant' is the important point in most instances, and this should not be difficult except possibly in the case of proliferative mastitis. Here the epithelial cells are very numerous indeed. They are small, deeply stained, and arranged in placards, but it may not be easy to see the cell boundaries.

Table III

DISEASE	NO. OF CASES	WET FILM DIAGNOSIS INACCURATE
Chronic mastitis Atrophy of breast Galactocoele Hæmatoma	41	1 diagnosed as carcinoma. The case was actually one of proliferative mastitis
Tuberculosis	3	None
Fibro-adenoma	17	7—4 cases were diagnosed as carcinoma 3 cases were diagnosed as duct papilloma
Fibroma	3	1 diagnosed as a giant-cell sarcoma
Duct papilloma	9	None
Sarcoma	5	1 diagnosed as an inflamed fibroma
Carcinoma	134	2— <i>a</i> "No evidence of malignancy" When re-examined this was obviously wrong and many carcinoma cells were found in the wet film <i>b</i> "No evidence of malignancy" The films were made from the wrong place

The fact that a galactocoele may simulate tuberculosis both clinically and microscopically was emphasized by the Royal Commission on Tuberculosis. In wet films the special features of the two conditions are clearly demonstrated, and in this series a correct diagnosis has been possible in every case. It will be remembered that chronic inflammation is in evidence in both conditions, but in a galactocoele there is usually some remnant of acute inflammation, and to this are added the typical foreign-body giant cells which are often filled with lipoids. In tuberculosis endothelial cells may be present in large numbers. Foam cells are found in lesions of the breast such as chronic inflammation or mastitis, and it is unusual to discover them in wet films made from malignant growths unless the scraping has included mastitis around the growth or an area of lactating tissue.

The main point, however, is the nature of the carcinoma cells and their relation to the type of cell found in fibro-adenomata.

It has been noticed that there are three different and distinct types of carcinoma of the breast as presented in wet films. These types depend upon the actual appearance of the malignant cells and are not concerned with the clinical aspect of the tumour.

Type I—In this type the carcinoma cells are very large indeed. Their nuclei are often enormous and the cytoplasm of each individual cell is plentiful. This cytoplasm has a tendency to stain somewhat more pink with eosin than is usual. The cells are arranged in small groups and as isolated units, but there is never any indication of the tessellated appearance of benign placards. In addition multinuclear malignant cells are conspicuous, and these often show phagocytosis of red blood-corpuscles. A diagram of this type of carcinoma was given in the original paper¹

Type II—The cells are smaller than those of *Type I*, but are still considerably larger than normal. They have very little cytoplasm and are almost entirely separate from each other, this lack of epithelial formation is suggestive in some cases of a sarcoma. It is in this type that mitosis is especially obvious and the capillaries are sometimes very prominent.

Type III—The cells are small and of about normal size. They are very numerous and arranged in placards, the cell boundaries, however, cannot be seen. There is often evidence of invasion of fat and isolated cells are present.

It is the third type of carcinoma which has led to difficulty in distinguishing between carcinoma and duct papilloma or fibro-adenoma, for in all three the size of the cells is somewhat similar. It will be seen that in a total of 17 fibro-adenomata 4 were judged to be carcinoma of the small-cell type. Having realized that this type of carcinoma is found in wet films one is very much less likely to make the mistake, for the arrangement of the cells in the benign conditions is regular and even, whereas in the carcinoma the nuclei are unevenly spaced and lie upon each other in a characteristic fashion. Corroborative evidence may be found in the presence of isolated cells invading the fat. In a wet film the fat cells tend to lie in plaques like other benign cells and hence the presence of any malignant infiltration is quickly seen.

The significance of the three types of carcinoma cell is difficult to determine, and it was hoped that some light might be thrown upon the problem by studying the after-history of the patients. It has been possible to investigate 129 out of 134 patients, and of these 44 are dead already.

Although the time since operation is small in many of the cases, there is no evidence that any one of the above types is more lethal than another. The patients with the small-cell tumour die or recover in about the same proportion as the others.

With this follow-up available the question of the prognostic significance of mitosis was also investigated, with the same negative result.

Female Genital System—The results of investigations by wet films of lesions of the female genitalia have been studied and reported upon in detail by Wrigley.⁷

A point which might be of use in the early detection of pathological processes in the mucosa of the cervix uteri has been described in a paper by Schiller.⁹ It is important to be able to differentiate easily between normal and pathological conditions of the cervix, and it was found that the normal epithelium in this region contains glycogen in its superficial layers, and this glycogen can be demonstrated in the living patient by painting the area with Lugol's iodine solution (iodine, 1, potassium iodide, 2, water, 300). In pathological conditions of the mucosa, the absence of glycogen leaves the cells unstained by the iodine. The diagnosis between leucoplakia and carcinoma in the unstained areas could probably be settled by taking a scraping and making wet films.

MISTAKES AND DIFFERENCES IN DIAGNOSIS

A critical investigation of the mistakes in diagnosis which have been made in this series confirms the view that with increasing experience these should be reduced to a negligible figure. In the first 200 cases reported by Dudgeon and Patrick there were 9 instances in which the interpretation of the wet films was wrong, and these mistakes have been fully explained in their paper. In the remainder of the series there were only 20 mistakes. The wet films were made from the wrong part of the specimen in 5 of these cases, since the interpretation of the films as they were represented was undoubtedly correct and yet it differed from the section diagnosis. A typical example of this group was a film made from a tumour of the breast in which there was no evidence of neoplasm, but the section showed a cellular carcinoma.

In two films, the one made from a carcinoma of the prostate and the other from a lymphosarcoma of the subcutaneous tissues, a diagnosis should not have been attempted, since the preparations were so bad that the cells could not be recognized distinctly.

Three cases were correctly diagnosed at a second examination of the films, and it is admitted that these mistakes should not have occurred.

The difficulty of differentiating between fibro-adenoma of the breast and the small-cell type of carcinoma has been explained elsewhere.

Two cases of endothelioma of lymph-glands were wrongly interpreted, the first being mistaken for lymphadenoma, although the film was very atypical of this condition, and the second for chronic lymphadenitis.

A diagnosis of carcinoma of the vas deferens was made on one occasion in a case in which the testicle had been removed for gonococcal epididymitis. The inflammatory reaction in the walls of the vas was responsible for the unusual appearance of the epithelial cells.

Finally there were two cases in which the diagnosis given from the film cannot be explained and the mistake would probably be made again until further experience is available. The first film was made from a hæmangioma of the neck in which the cells seemed to show the features of a carotid body tumour, and the second from a ganglion of the palm which was undergoing organization, and this was thought to be a myxosarcoma.

In the whole series there were 17 occasions on which a section was not made from the material used for the wet film. These were mostly cases in which a 'rapid diagnosis' was given in the Out-patient Department or the operating-theatre, and since in this group there is definite evidence available that the diagnosis was correct, the numbers have been included.

Summarizing these results, it is evident that the number of cases in which an inaccurate diagnosis was given is extremely small, and the majority of these can be accounted for by errors in technique.

It remains to be said that there are many in which a more valuable and complete diagnosis was possible from the wet film than from the corresponding section. This occurred most frequently in the diagnosis of tuberculosis, which was often indefinite or doubtful in the sections and yet the picture was quite typical in the corresponding films. It is also a fact that in 8 cases a diagnosis of malignancy was made from the film, and although this was not demonstrated by the section

at the time, the fate of the patients confirmed the opinion. These cases are shown in *Table IV*

Table IV

ORIGIN OF SPECIMEN	WET-FILM DIAGNOSIS	SECTION DIAGNOSIS
Bone	Giant-cell sarcoma	Active new bone formation in and around fibrous myositis
Bladder	Carcinoma	No tumour present
Stomach	Carcinoma	No evidence of carcinoma
Tumour of leg	Spindle-cell sarcoma	Necrotic material
Uterus	Carcinoma	No evidence of malignancy
Thyroid	Carcinoma	Fœtal adenoma
Kidney	Carcinoma	Papilloma
Bladder	Carcinoma	Papilloma

SUMMARY

1 The method of examination of new growths and inflammatory diseases by wet films has been extended and applied to pathological processes in general

2 Over 1000 cases have been investigated and the features of interest are reported upon and reviewed

3 The application and technique of the method are described in detail

4 The characteristic appearances of benign and malignant conditions are considered and the differential diagnosis is discussed

5 The special histological features of various types of malignant cells are described and the suggestion that there is a definite difference between benign and malignant cells is put forward

6 The finding of malignant cells in tissues which are apparently normal in appearance and at some distance from the original focus of disease has been possible in a large percentage of the cases examined with this object in view

7 The possibility of early diagnosis of malignant change in benign growth such as papillomata of the bladder is recorded

8 The errors in diagnosis which have been made are recorded and reference is made to the particular pitfalls which may be expected

9 A number of instances are given in which the diagnosis suggested from the wet film was more complete or more accurate than that of the corresponding section

REFERENCES

- ¹ DUDGEON, L. S., and PATRICK, C. V., "A New Method for the Rapid Microscopical Diagnosis of Tumours, with an Account of 200 Cases so Examined", *Brit Jour Surg*, 1927, *xv*, 250

- ² SHAW, E H, "The Immediate Microscopic Diagnosis of Tumours at the Time of Operation", *Lancet*, 1910, Sept 24, *Ibid*, 1923, Feb 3
- ³ SHATTOCK, S G, and DUDGEON, L S, *Proc Roy Soc Med* (Pathol Sect), 1914, Dec 1
- ⁴ WENYON, C M, *Protozoology*, 1926, 11, 1321
- ⁵ BLAND-SUTTON, Sir J, *Tumours Innocent and Malignant*, 7th ed, 1922, 258
- ⁶ LUDFORD, R J, "The Differential Reaction to Trypan-blue of Normal and Malignant Cells 'in vitro'", 10th Scientific Report, Imp Canc Res Fund, 1932, 169
- ⁷ WRIGLEY, A J, "A Method of Rapid Diagnosis of Pathological Specimens", *Jour Obst and Gynæcol Brit Emp*, 1932, xxxix, 527
- ⁸ CARLETON, H M, "Studies on Epithelial Phagocytosis", *Proc Roy Soc, B*, 1931, cviii
- ⁹ SCHILLER, W, "Early Diagnosis of Carcinoma of the Cervix", *Surg Gynecol and Obst*, 1933, lvi, 210

RECONSTRUCTION OF THE FOREARM AFTER LOSS OF THE RADIUS

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THE alluring prospect of healing by first intention in acute osteomyelitis provides a strong temptation to the surgeon to practise the operation of diaphysectomy. Unfortunately the early removal of the only available source of calcium and the collapse of the periosteal tube so commonly result in failure of regeneration that the operation cannot be performed with safety except in osteomyelitis of the fibula. In osteomyelitis of the radius, even with less destructive operations, there is a strong tendency to the development of radial club-hand with dislocation of the lower end of the ulna, and if the whole shaft of the bone is removed, the complication is almost inevitable (*Figs 7, 8*).

The resulting deformity cannot be adequately treated by a bone-grafting operation. The difficulty lies not so much in the bridging of a wide gap filled with



FIG 7—Failure of regeneration of the radius following diaphysectomy for osteomyelitis. Condition of the limb when first seen twelve months after original operation.

scar tissue between two very small fragments of bone, but in the preliminary replacement of the distal fragment of the radius in normal relationship to the ulna. If the inferior radio-ulnar dislocation is not reduced, the ugly prominence of the lower end of the ulna remains, and satisfactory radio-ulnar movement cannot be restored.

The operation described by Hey Groves¹ of implantation of the distal end of the ulna into the small distal fragment of the radius offers a solution to the problem, and this case is reported to illustrate the excellent cosmetic and functional result which may be anticipated from the construction of a single forearm bone.

The patient was a girl of 19 years, and diaphysectomy of the radius had been carried out by another surgeon twelve months before she was first seen. The old scar was excised, the lower end of the ulna dissected out subperiosteally, and angulated into a drill-hole in the fragment of the radius. Almost the whole length of the ulna was preserved in order to minimize the shortening of the limb, and the fragments were impacted in a position of 10° pronation from the mid radio-ulnar position (*Fig 9*)



FIG 8—X-ray of forearm before reconstruction operation

Although this construction of a single forearm bone involves complete loss of radio-ulnar movement, it is easy by internal rotation and slight abduction of the shoulder to bring the hand into the full palm-down position when resting on a table (*Fig 10*), or by adducting and externally rotating the shoulder to turn the palm of the hand towards the face or the back of the neck (*Fig 11*). Wrist movement has recovered to 80 per cent of normal, and it is not possible without a very careful examination to know that there is any abnormality of the limb



FIG 9 —X-ray of forearm two years after Hey Groves's operation (construction of a single forearm bone)



FIG 10 —Photograph showing the excellence of the cosmetic result

In 1932 Greenwood² described a modification of the operation, he divided the lower shaft of the ulna, overlapped the two ulnar fragments, and impacted the shaft into the small radial fragment, leaving the lower end of the ulna as a buttress "to give greater stability to the wrist-joint" Such a buttress develops, however, from subperiosteal bone formation even when the original technique is followed



FIG 11—Result of operation, showing that there is no functional disability

(see Fig 9), and the wrist-joint acquires normal stability Greenwood's modification is unnecessarily complicated, it involves undue shortening of the limb, and leaves the disfiguring prominence of the lower end of the ulna, so that the cosmetic result is definitely inferior to that which may be secured from Hey Groves's original operation

REFERENCES

- ¹ GROVES, E W HEY, *Modern Methods of Treating Fractures*, 2nd ed, 320 Bristol
John Wright & Sons Ltd
² GREENWOOD, H H, *Brit Jour Surg*, 1932, 22, 58

LOCALIZED HYPERTROPHIC ENTERITIS AS A CAUSE OF INTESTINAL OBSTRUCTION WITH A REPORT OF TWO CASES

By W A JACKMAN

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A REMARKABLE coincidence, with which most surgeons are familiar, is that the incidence of one rare and extraordinary case is so often followed closely by that of another of similar character. When the first of the two cases recorded below came under my notice I was so much puzzled to explain its nature that I was inclined to try to forget it, but when, within eighteen months, a second patient suffering from a precisely similar condition presented herself, I felt that a careful description and drawings should be prepared, so that others might have an opportunity of criticizing or explaining.

CASE REPORTS

Case 1—E S, female, aged 37 years, married, was admitted to the Bristol Royal Infirmary on Aug 1, 1931, and gave the following history.

HISTORY—Twenty-four hours before admission she was suddenly seized with acute abdominal pain of a colicky nature, localized from its commencement to the right iliac fossa. This was followed by nausea and vomiting, and she had been unable to retain anything taken by the mouth. The bowels had not acted since the attack started, but she had suffered from some degree of constipation for years, which was easily relieved by mild aperients, otherwise she had been previously healthy and had not had any similar attacks of pain. The temperature was 99.6, the pulse 90, her tongue was dry and furred brown.

ON EXAMINATION—The positive findings were tenderness, rigidity, and guarding in the right iliac fossa. The customary additional examinations yielded negative results and there was no palpable tumour.

The diagnosis of acute appendicitis, made by her own doctor, was confirmed, and operation immediately was advised and undertaken.

OPERATION—The approach was made through a gridiron incision on the right side, and on digital exploration of the peritoneal cavity in the appendix area a section of bowel was felt which, to the touch, gave the impression of being an intussusception. The affected segment was easily identified as terminal ileum. The cæcum, appendix, and abnormal segment of bowel were delivered on to the abdominal wall. Although this delivery was effected with care and gentleness, it was apparent that the manipulation had resulted in perforation of the bowel wall, and it was this accident which determined the procedure eventually adopted.

The affected bowel, about 8 in. in length, was intensely congested, in parts almost black. It felt thickened, yet on handling proved to be fragile, there was no intussusception and no constriction ring. The commencement of both the congestion and thickening was sharply defined and terminated abruptly at the ileocæcal junction, the cæcum and appendix appearing normal. There was no evidence of thrombosis of the mesentery. On account of the diseased bowel having perforated, it was thought inadvisable to return this to the abdomen or to make any further encroachment upon the peritoneum, and a local excision was performed, the incision of the mesentery being made close to the bowel, in order to

preserve an efficient blood-supply to the remaining gut and to the anastomosis. The anastomosis was made between the end of the remaining ileum and the side of the cæcum, from which the ileocaecal valve had been removed, and was most difficult to accomplish. It was necessary to remove the appendix, as its mesentery prevented access to the cæcum.

A safety-valve drain, in the form of a catheter, was stitched into the anterior part of the junction line, and projected into the lumen of the ileum for about two inches, and finally the peritoneum around the aperture so made was stitched to the peritoneal edges of the incision. The remaining peritoneum, muscle layers, and skin were closed in the usual manner, the catheter being held in place by one silk-worm-gut stitch to the skin edge.

SUBSEQUENT PROGRESS—Three days after the operation the patient developed a sore throat which proved to be diphtheria, and, the surgical condition being considered satisfactory, she was removed by ambulance to the city isolation hospital. While there she developed paralytic complications, but in spite of this her surgical condition progressed favourably, and the faecal fistula resulting from the drainage of the bowel closed rapidly after removal of the drain. The diphtheria and its complications terminated satisfactorily, and she was discharged, cured of all her ailments, some three months after admission.

PATHOLOGICAL REPORT—The resected portion of bowel was examined in detail in the pathological laboratory attached to the hospital, and the report received reads as follows—

The specimen is a portion of small bowel about 15 cm. in length. The wall is much thickened in most of its extent and measures 2 cm. at the thickest part, near the distal end, where the lumen is much narrowed. Near the proximal end the lumen becomes moderately dilated, but the bowel wall shows no thinning. The peritoneal surface is markedly congested, smooth and glistening and shows no surface exudate. No thrombosed vessels can be seen.

The mucosa, over a very wide area, has been entirely replaced by a firm hæmorrhagic exudate, with roughened surface. At the proximal end the margin of this mucosal ulceration forms a clearly defined but irregular line. Although the healthy mucosa beyond shows some congestion, the transition from the diseased to the healthy segment is somewhat abrupt. Near the centre of the ulcerated mucosal surface and on the mesenteric side, the bowel wall is thinned out to a small ragged-edged opening, representing a more severely ulcerated area which has been further damaged during the removal of this part of the bowel.

The microscopical examination reveals a condition of simple intestinal ulceration. The mucosa throughout the affected area has been completely destroyed and replaced by thick fibrino-purulent exudate. The only bacteria demonstrated in the ulcerated areas are those of the intestinal tract group, and of these streptococci are the predominating ones. There is no histological evidence of any of the granulomatous infections, and nothing in the nature of new growth can be found.

In the deeper layers and throughout the rest of the bowel wall no bacteria of any sort can be seen. In the bowel wall generally there is a marked acute inflammatory reaction, lessening in degree towards the subperitoneal layer, which shows congestion, œdema, and interstitial hæmorrhage but few polynuclear cells. This is in marked contrast to the condition in the submucosal and muscle layers, where, in places, the degree of polynuclear cell infiltration approaches abscess formation.

Evidence of previous acute inflammation of the bowel is present in the form of much dense scar tissue. There is much fibrotic thickening of the submucosa, and extensive areas of the muscle coat have been entirely replaced by fibrous tissue. Beyond a very occasional small vessel there is no evidence of vascular thrombosis.

The accompanying excised appendix is normal, and on section shows no evidence of inflammatory change.

Case 2—I. T., female, aged 25 years, unmarried. Admitted to the Bristol Royal Infirmary on Jan. 1, 1933, complaining of acute abdominal pain which had commenced forty-eight hours before admission and was localized to the right iliac fossa during the whole of this period. Vomiting was a prominent symptom, and she had not been able to retain anything taken by mouth.

HISTORY—The patient's previous history revealed the fact that since childhood she had suffered from mild dyspepsia, not constant or sufficiently severe or continuous to demand medical attention, and never accompanied by vomiting, and that she had always had severe dysmenorrhœa of the congestive type. She was not menstruating at the time

of admission. The bowel action had always been regular with a slight tendency to diarrhœa, which she never regarded as of any importance, so little inconvenience did it cause. She had never had an attack of pain similar to that from which she was suffering on admission.

ON EXAMINATION —The temperature was 100° and the pulse 96, the tongue furred brown, and dry. The positive findings on examination were similar to those in *Case 1* and no tumour was felt in the abdomen. It was thought that she was suffering from acute appendicitis, the diagnosis made by her own doctor and urgent operation was advised, to which she consented.

OPERATION —The same method of approach was used as in the operation just described, the results of the local exploration and delivery of the diseased bowel exactly correspond, even to the perforation of the gut on gentle manipulation. The appearance of the lesion, its distribution, consistency and colour left no doubt that the condition was the same as that encountered in the previous case, and again the extent of the operation was limited by a perforation. The difficulty of performing efficiently an anastomosis by the method adopted before led to a modification in the technique, which was as follows. The diseased segment of gut was excised and the terminal end of the proximal ileum closed in the manner ordinarily adopted in intestinal resection, as also was the cæcum at the site of the excised ileocæcal valve. A side-to-side anastomosis was carried out, ileum to anterior cæcal wall. The gut was not drained. This modification rendered the operation easy, and would be adopted by the writer without hesitation if a similar condition should ever be encountered again.

SUBSEQUENT PROGRESS —The patient made an uninterrupted recovery, except that at one small area in the scar some hypertrophic granulation tissue, which did not yield to the application of caustics, required to be cut away. This area corresponded to the site of a small rubber dam drain which was left in the incision down to peritoneum for the first three days after operation. Although convalescence was uncomplicated, it was slow, and the patient was not able to return to her work, domestic employment, until five months after the operation. She is now well, but volunteers the statement of two interesting facts: (1) That whereas before the operation her bowels were inclined to be loose she now has to take laxatives occasionally or she becomes constipated, and (2) That her dysmenorrhœa is absolutely cured.

PATHOLOGICAL REPORT —The pathologist's report on the specimen removed is almost word for word that given on *Case 1*, with the minor exceptions that the length of the excised bowel in this case is 20 cm. and the bacteria present do not include streptococci and are comparatively scanty.

The illustrations are taken from this case, *Fig. 12* represents the appearance of the diseased bowel at the time of operation. *Figs. 13, 14* are copied from the actual specimen, and the microphotographs (*Figs. 15, 16*) are also taken from sections cut from the same preparation.

COMMENTARY

The writer has not been able to discover in the surgical literature available an account of any parallel case, and the diseases to which the ileum is liable do not correspond in any instance with the condition found in these patients.

Apart from the interest aroused by the discovery of so massive a lesion with so short a history, at any rate of severe symptoms, the questions of nomenclature, diagnosis, and treatment arise. It is a difficult task to name appropriately a lesion which shows such a variety of diverse pathological changes without any obvious causative factor. At first sight the appearance of deep congestion in the gut and its sharply limited extent strongly suggest that there has been some strangulation. An internal hernia, strangulation by a band, or a volvulus might each cause this appearance of abruptly limited strangulation, or a thrombosis of one of the terminal branches of the ileocolic artery might have produced the same apparent result. But even if we ignore the fact that neither incarceration, twisting, thrombosis, nor strangulation by band were found, we are confronted by two facts which prove that the causative condition must have been of a somewhat chronic nature. The

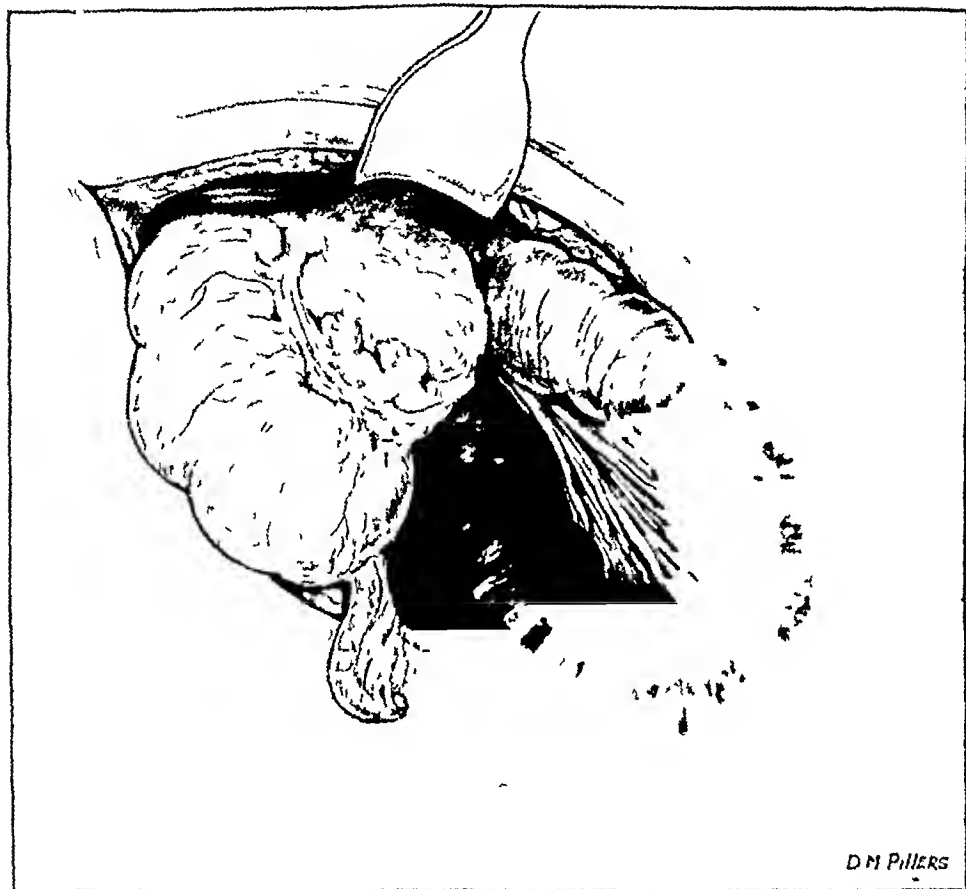


FIG 12—Condition of ileum found at operation. Note abrupt commencement and termination of inflammatory lesion

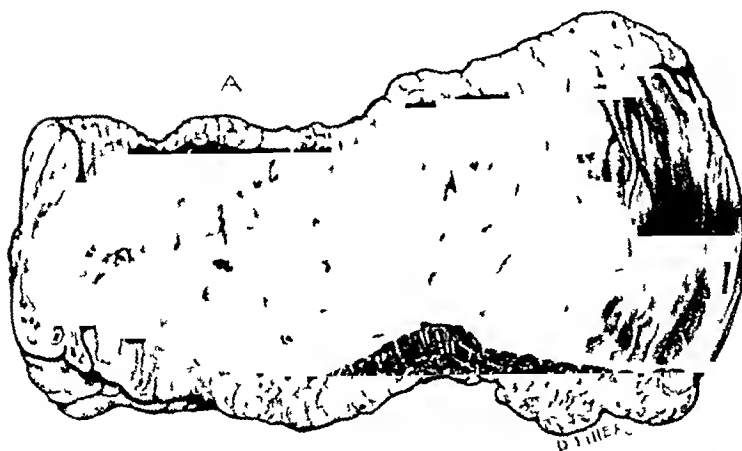


FIG 13—Portion of ileum removed, opened by longitudinal incision, showing the intense congestion of the mucous surface, extreme fibrous thickening of the intestinal wall. Note extensive necrosis of small area and perforation (A)

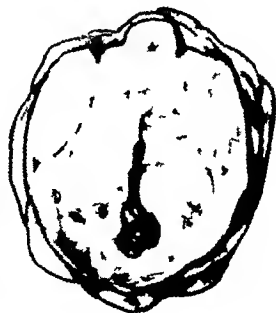


FIG 14—Transverse section to show thickened wall of ileum and obliteration of lumen

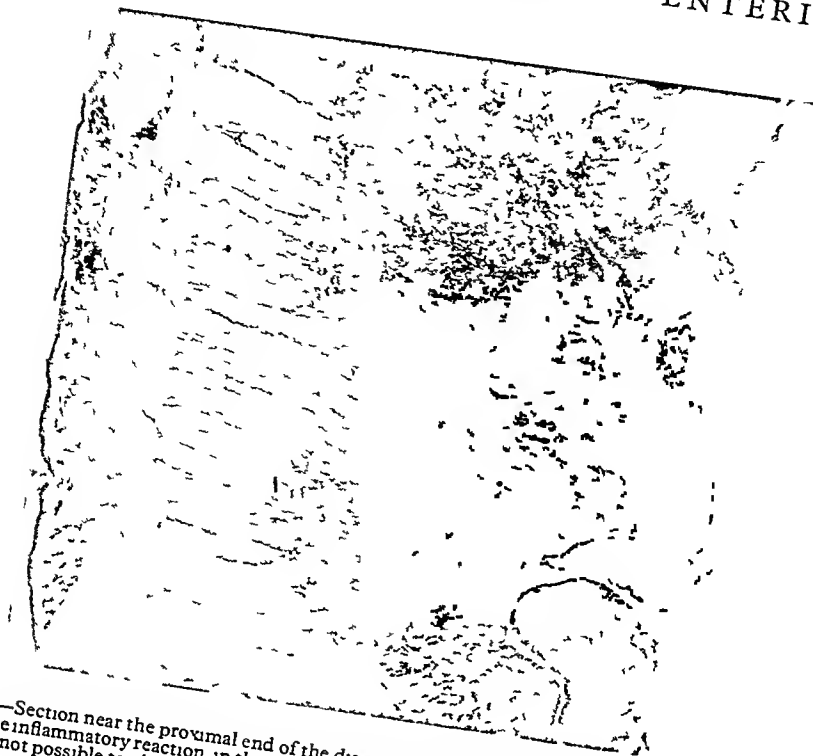


FIG 15 —Section near the proximal end of the diseased portion of bowel to show the ulcerated mucosa and the intense inflammatory reaction in the submucosa. Shows also extreme thickening, but for technical reasons it was not possible to obtain a full thickness' photograph at the most thickened portion (x 20)



FIG 16 —Section through the junction between diseased and healthy bowel. The space is due to contraction of the tissues during preparation of the section (x 90)

first of these is that there is enormous thickening of the bowel wall, consisting of well-formed fibrous tissue, and the second that the mucous membrane has been almost completely destroyed by ulceration. The main change seems to be that of hypertrophic reaction to an inflammatory process which is mainly in excess of the destructive ulceration present. 'Subacute hypertrophic ulcerative enteritis' is the designation suggested.

It will be noticed that the symptoms and signs are not quite typical of acute appendicitis, and to cite one point which did suggest some other condition, the pain commenced in and remained in the right iliac fossa. There are many who will agree that this fact alone should always demand an exhaustive search for some other lesion. In the present instance one might have expected the discovery of a sausage-shaped tumour on palpation of the abdomen, a sign which was not elicited, probably on account of the rigidity of the abdominal muscles on the affected side.

The most common positions in which the appendix is situated are the retro-cæcal sites and the pelvic situation, the splenic, inguinal, and promonteric positions being comparatively rare. When inflamed and in the first-mentioned position loin tenderness can almost always be elicited, and in the pelvic site tenderness in the pouch of Douglas. These signs were absent in the present instances.

It is suggested that the diagnosis of the disease described here might be borne in mind in cases presenting atypical features in history or signs of acute appendicitis, and that considerable effort should be directed to the finding of a tumour which should be palpable.

In regard to the treatment it may be as well to consider alternatives first. The diseased gut might have been left *in situ* in the hope that it would undergo spontaneous cure. This seemed, and still seems, to be taking a graver risk than that of active intervention, the issue is impossible to determine without the benefit of the experience of others who, faced with like circumstances, have adopted the conservative course.

Then again, a more radical and orthodox operation might have been performed, which would actually have been easier and would have assured vascular stability in accordance with recognized anatomical considerations.

In defence of the procedure adopted it can be said that the blood-supply to the remaining bowel was never in any doubt. Before the anastomosis was commenced the ileum and cæcum were watched for several minutes, in order to determine the fact that they possessed a blood-supply sufficiently efficient to promote the necessary healing of the anastomotic line.

The object of all treatment, medical or surgical, is to restore our patients to their normal physiological balance and, as far as possible, to preserve their designed anatomical conformation with the object of the restoration of normal health. This object has apparently been attained by the method defined here.

I am indebted to Dr A. D. Fraser, who has so carefully prepared the pathological reports and the microphotographs and has taken infinite pains to elucidate the lesion and to discover a similar condition in the literature of pathology. My thanks are due also to Professor E. W. Hey Groves for his interest and help in many ways, to Mr C. F. Walters and Mr A. W. Adams at whose request the cases came under my care, and to Miss D. M. Pillers, who has so faithfully reproduced in the sketches the features which I desired to demonstrate.

GASTROJEJUNAL ULCERATION

By A JAMES WALTON

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OUR experience of the nature and frequency of gastrojejunal ulceration is of necessity short, for the first gastro-enterostomy was performed by Wolfier, at the suggestion of his assistant Nicoladoni, as recently as the year 1881. Possibly because the earlier operations were generally performed for carcinoma of the pylorus, the first case of gastrojejunal ulcer was not reported by Braun⁹ until 1899, this being one in which a fatal perforation took place. In the short period of thirty-five years that has ensued since this date our knowledge has been steadily increasing. At first only single cases were reported at long intervals, the first in France being reported by Quenu in 1902, and the first in this country by Mayo Robson in 1904. After this they were found in rapidly increasing numbers, Paterson,³⁹ in his epic paper in 1909, first directing widespread attention to the subject, although he found that Brodnitz¹⁹ in 1903 was able to collect 15 cases, Gossett¹⁷ in 1906 31 cases, and Einer Kay¹³ a year later added 6 examples. Paterson in his own paper collected 52 certain and 11 doubtful cases, 2 of which were from his own practice.

Of later years so many examples have been reported that it is no longer practical to collect individual cases but only to consider general statistics. So many indeed have been reported that a keen controversy has arisen as to the actual frequency of the condition. On the one hand, Lewisohn²⁸ in 1925 published a series of 68 cases of gastro-enterostomy where 23, or 34 per cent, developed gastrojejunal ulceration after having been watched for a period of not less than five years. Hurst and Stewart²⁴ believe that it is a dangerous and frequent sequel of gastro-enterostomy. They cast doubt upon the accuracy of many of the figures reported by various surgeons, and claim that the number of anastomotic ulcers slowly but steadily multiplies as the cases of gastro-enterostomy are watched over increasing periods. They give pathological statistics showing a 73 per cent incidence in 42 cases examined at Leeds, ten days to two months after operation, and a 52 per cent incidence in 42 cases examined nine months to nineteen years after operation. These, however, must be considered as very selected cases, for the examinations were carried out on patients who had died, and therefore took no cognisance of those who had recovered and might have formed a high percentage of the total operated upon. These extreme views as to the frequency of this dangerous complication are held by a considerable number of surgeons of wide experience. This is especially so on the European continent, where many have abandoned the routine use of gastro-enterostomy and have replaced it by some form of partial gastrectomy. Among those who hold these views may be quoted Finsterer,¹⁴ von Haberer,⁴⁷ Hohlbaum,²² von Eiselsberg,⁴⁴ and in this country Pannett.³⁸

On the other hand, many surgeons, and especially those in this country and the United States, have found that recurring ulceration of this type, although

dangerous and not entirely unavoidable, occurs in only a small percentage of the cases, and is insufficiently frequent to warrant the abandonment of so useful an operation as gastro-enterostomy. Paterson in 1909 found only 3 gastrojejunal ulcers in his own series of 348 cases, and in a later paper found 12 examples in 495 cases—that is, 2.4 per cent. Lord Mownihan¹⁷ in 1919 found 10 cases in 613 gastro-enterostomies, or 1.6 per cent. Balfour² found that a recurrent ulcer developed in only about 3 per cent of the gastro-enterostomies performed for duodenal ulcer. In a series I published in 1925¹⁸ there were 631 cases of gastro-enterostomy with 11 gastrojejunal ulcers—that is, 1.7 per cent. Of these, 417 were performed for pyloric and duodenal ulcers with 10 recurrent ulcers—that is, 2.4 per cent. In a later series published in 1930¹⁹ there were 616 gastro-enterostomies, 16 subsequently developing gastrojejunal ulcers—that is, 2.6 per cent. These figures agree very closely with those published by Luff¹⁰ from the collective investigation of the British Medical Association, where, of 995 cases of duodenal ulcer, 744 were traced, of these 21 (2.8 per cent) had a subsequent history of gastrojejunal ulcer.

Many of the published figures are, however, depreciated in value by the fact that no clear distinction is made in the statistics between cases operated upon for gastric or duodenal ulcer. Moreover, the criticism is not infrequently made that the cases have not been sufficiently carefully followed or have been watched for too short a time. In this respect it may be important to remember that physicians are likely to see only the surgical failures, just as the surgeons are only asked to treat patients resistant to medical treatment, and thus each is prone to believe that there is a much higher percentage of such failures than really exists.

It was in the belief that light might be thrown upon some of these much discussed points that the Association of Surgeons undertook a collective investigation into the subject of gastrojejunal ulcer. The subject matter of the present communication is based upon a study of my own cases prepared for this investigation.

TOTAL INCIDENCE

By the courtesy of the London Hospital authorities I have been able to establish a Follow-up Department which has been in existence since 1919. In this department all cases are examined at intervals which at first are quite short and later are increased as the patient remains well. This has been so successful that I feel confident that no case of gastrojejunal ulcer has escaped my notice, although several have been treated and operated upon elsewhere, a record has always been procured. I can therefore feel that the figures quoted are reliable and are as accurate as can be obtained for any series of cases under the care of one surgeon.

For the collective investigation it was decided to consider only the cases which were operated upon in the years 1926–30 inclusive. The frequency of gastrojejunal ulceration in this group is shown in *Table I*, and it is seen that the incidence of this complication was 0.73 per cent. It was realized, however, that these figures are considerably below those which I had previously published, and it was thought that this might be due to the fact that the series contained too few cases and that a more correct estimate might be obtained by a study of all the cases which had occurred in my practice. These figures are shown in *Table II*, and it will be seen that, excluding 18 cases of gastrojejunal ulceration where recurrence followed a local operation, there were 31 gastrojejunal ulcers following 1859 gastric operations.

—that is, an incidence of 1.6 per cent. One of these cases was a recurrence after a partial gastrectomy for a gastrojejunal ulcer, so that there were really only 30 cases of fresh marginal ulceration.

Table I—CASES OPERATED UPON 1926–30

	TOTAL	M	F	TRACED	GASTRO- JEJUNAL ULCER	PER CENT
Duodenal ulcer	319	252	67	All	4	1.22
Gastric ulcer	254	167	87	All	—	—
Gastric carcinoma	71	44	27	All	—	—
Gastrojejunal ulcer	32	28	4	All	1*	3.10
Totals	676	491	185	All	5	0.73

* Recurrent

Table II—ALL CASES

	TOTAL	M	F	TRACED	GASTRO- JEJUNAL ULCER	PER CENT
Duodenal ulcer	893	737	156	All	29	3.24
Gastric ulcer	683	446	237	All	1	0.14
Gastric carcinoma	221	148	73	All	—	—
Gastrojejunal ulcer	62	58	4	All	1*	1.60
Totals	1859	1389	470	All	31	1.60

	TOTAL	M	F	TRACED	ULCER
Gastrojejunal ulcer (other operations)	18	15	3	All	6*

* Recurrent

Those who believe with Hurst²⁴ that the cases increase in number the longer the patients are followed will regard this increased incidence as due to the inclusion in the list of many cases operated upon before 1926. This is in part true, but it is not the sole explanation. My own cases date back to 1913. In 1923 the statistics were carefully investigated and published, and the present series was prepared to include cases up to September, 1933, so that the two ten-year periods can be easily compared. In the ten years, 1913–22 inclusive, there were 765 gastric operations, and the present investigation shows that of the 30 patients who had gastrojejunal ulcers 18 were operated upon in this period, giving an incidence of 2.3 per cent as compared with the total of 1.6 per cent.

THE NATURE OF THE PRIMARY LESION

It is now a well-recognized fact that this complication is more prone to follow operation for certain types of ulcer than for others. Paterson³⁹ had noted this in his paper in 1909, when he stated that 33 of the certain 52 cases followed operation

performed for pyloric stenosis, and found only 1 case, that reported by Einer Kay, following gastro-enterostomy performed for gastric carcinoma Hurst²⁴ quotes Judd²⁶ as having likewise known, up to 1921, only 1 case occurring after operation for gastric carcinoma. He also states that it occurs but rarely in cases of gastric ulcer, but is a frequent sequel after duodenal ulcers. These statements are well supported by the present series (*Tables I and II*), for it will be seen in *Table II* that, whereas 29 ulcers occurred after 893 operations for duodenal ulcer (3.24 per cent), there was only one after 683 operations for gastric ulcer (0.14 per cent), and not a single example after 221 operations for gastric carcinoma. The highest incidence therefore is among duodenal ulcers. The figures for the cases operated upon in 1913-22 inclusive give 457 gastro-enterostomies for duodenal and pyloric ulcers, with 18 gastrojejunal ulcers, a maximum incidence of 3.9 per cent, all of these cases having been followed for a period of over ten years.

The actual number of cases of gastrojejunal ulcer included in this investigation is 79, for in addition to the 30 cases occurring in those patients where I had personally operated there were 49 in whom the primary operation was performed elsewhere. In these 49 cases there was only 1 in which it could not be certain that the first operation was performed for a pyloric or duodenal ulcer. It may be accepted, therefore, that gastrojejunal ulceration is a complication almost entirely limited to gastro-enterostomy performed for ulcers in the neighbourhood of the pylorus or duodenum. After operations for gastric ulcer or carcinoma the condition is so rare as to be negligible.

THE VARIETY OF PRIMARY OPERATION

Although marginal ulceration is more common after operations for duodenal ulcer than for other lesions, there is evidence that the incidence varies according to the nature of the anastomosis that has been performed. Even in his early paper Paterson stated that it was believed to be more common after anterior than posterior gastro-enterostomy, for there were 27 anterior anastomoses, 10 anterior with a lateral anastomosis, 2 'in Y', 12 posterior, and 1 supracolic. He believed that the apparently greater frequency after the anterior operation was due to the fact that with it a lateral anastomosis was more often performed, and therefore, as also after the 'in Y' operation, the acid was not neutralized.

Lord Moynihan³⁷ in 1919 found 6 examples after 189 anterior, and 4 after 444, posterior anastomoses. Wright also believes that the lesion is more common after the anterior anastomosis, for he found in his series that the anterior operation had been performed in 72 instances and the posterior in 59.

That the incidence is higher after the performance of a lateral or an 'in Y' anastomosis is almost certain, as will be shown later when the relation of such ulceration to hyperacidity is considered, but it is difficult to believe that it is more common after a simple anterior anastomosis. In my own series this has certainly not been the case (*Tables III and IV*). In *Table III* the cases operated upon in 1926-30 inclusive are alone considered, and whereas after 446 posterior anastomoses there were 4 gastrojejunal ulcers—that is, 0.89 per cent—there was not a single example after 33 anterior unions. In *Table IV* all cases are considered, and 1313 posterior anastomoses were followed by 29 gastrojejunal ulcers—that is, 2.2 per cent—but 77 anterior unions again showed no cases of marginal ulceration. In the 49 cases

where the operation had been performed elsewhere there were 43 examples following the posterior and 4 following the anterior union (*Table V*) In two of the latter a lateral anastomosis had been performed

Table III—CASES OPERATED UPON 1926-30

	TOTAL	TRACED	GASTRO- JEJUNAL ULCER	PER CENT
Posterior gastro-enterostomy	446	All	4	0.89
Anterior gastro-enterostomy	33	All	—	—
Anterior Polya	16	All	—	—
Posterior Polya	181	All	1	0.55
Totals	676	All	5	0.73

It is widely believed that the lesion does not occur after the operation of partial gastrectomy, and it is acting on this belief that certain surgeons, as already mentioned, have replaced gastro-enterostomy by some form of partial gastrectomy in the treatment of duodenal ulcer. A considerable number of such recurring ulcers have, however, now been described. Hogue²¹ and Cole²¹ reported the presence of a large marginal ulcer occurring five years after the performance of the Polya operation for a duodenal ulcer. Beer⁴ and Wright⁵⁴ also report cases, and the latter suggests that the popularly accepted belief that these ulcers are rare after partial gastrectomy

Table IV—ALL CASES

	TOTAL	TRACED	GASTRO- JEJUNAL ULCER	PER CENT
Posterior gastro-enterostomy	1313	All	29	2.20
Anterior gastro-enterostomy	77	—	—	—
Partial gastrectomy	469	—	2*	0.43
Other operations	1859	—	31	1.60
	313	—	6†	
Totals	2172	—	37	1.70

* One recurrent

† Recurrent

is probably due to the fact that this operation is most frequently performed for carcinoma of the stomach, following which a gastrojejunal ulcer is rare. Hurst²⁴ quoted Birgfeld⁵ as having collected 67 cases from the German literature, and states that he has himself seen reports of 14 cases published since then. Three cases had occurred at the New Lodge Clinic, 2 of which were performed for gastrojejunal ulcer. Haberer⁴⁶ reported a case occurring seven years after the performance of a Billroth II operation for duodenal ulcer, and for this reason returned to a modification of the Billroth I operation, where he closed the stump of the duodenum and implanted the cut end of the stomach into the anterior surface of the duodenum below the closed opening.

In my own series (*Table IV*) there were 469 partial gastrectomies, in 2 of which gastrojejunal ulcers followed—that is, 0.43 per cent. 143 of these operations, however, were performed for carcinoma, so that of 326 operations performed for ulcers 2 examples of marginal ulceration were seen—that is, 0.61 per cent. Of these two cases, one followed a Polya gastrectomy for a marginal ulcer occurring after a posterior gastro-enterostomy performed elsewhere, and was definitely due to a technical error, in that, owing to extensive adhesions, I was content to remove a smaller portion of the stomach than usual. A second more extensive operation was performed, and the patient has since remained well. The other was an acute ulcer occurring after a Polya resection, and as the operation was performed in 1915, a smaller portion of the stomach was removed than would have been taken to-day. In the 49 cases of marginal ulcer in my series in which the primary operation had been performed elsewhere (*Table V*), there were also 2 examples following partial gastrectomy, in both cases of the Billroth II variety.

Table V—GASTROJEJUNAL ULCERS

	TOTAL	M	F	DUODENAL	LESSER CURVE	P G E	A G E	BUTTON	IN Y	P G	SILENT ULCER
Self	30	29	1	29	1	29	—	—	—	2†	2
Others	49*	43	6	48	1	43	4	1	1	2	13

* In this group there were 51 operations in 49 cases, for two patients had recurrent ulcers

† One recurrent

There seems therefore no doubt that a partial gastrectomy does not give a complete immunity to the onset of gastrojejunal ulcer, but it is less frequent after this operation—about 0.5 per cent—than after gastro-enterostomy. My own belief is that, provided sufficient of the stomach is removed to produce an achlorhydria, the complication is almost unknown, although the risk of subsequent anæmia may be increased thereby.

From time to time much stress has been laid upon the fact that gastrojejunal ulcer does not follow gastroduodenostomy. This must of course be so. If there is no gastrojejunal anastomosis there can be no gastrojejunal ulceration. Its place would be taken by recurrent ulceration. A full discussion of the question of such recurrence is outside the scope of this paper, but it may be stated that Grey Turner,¹⁸ in a series of 43 such cases, found 4 recurrences.

THE RELATION OF MARGINAL ULCERATION TO OPERATIVE TECHNIQUE

It has frequently been suggested that the incidence of marginal ulceration is increased by technical errors in the operation. In the earlier cases it would seem probable that the use of mechanical devices, such as a Murphy's button or a bobbin, by causing necrosis of the opposed edges, instituted a process of ulceration. This might have progressed to a chronic marginal ulcer, and the inclusion of a large number of such cases might account for the exceptionally high incidence of this

lesion in some of the earlier series that have been reported. Garnett Wright⁵⁴ states that in his series of 155 collected cases a Murphy's button or some form of bobbin had been used in 14. In my own series there was no example in the 30 cases following my own primary operation, for I have never used any method other than a simple suture, but in the 49 cases following operation performed elsewhere there was one in which a Murphy's button had been used for the anastomosis after a perforated duodenal ulcer. The button was still in place and was associated with a chronic marginal ulcer.

At one time many surgeons, including myself, were of the opinion that the use of unabsorbent sutures was an important factor, however, the knowledge that such ulceration occurs when catgut has alone been used makes this position no longer tenable, but the presence of pieces of silk in the base of a large number of these ulcers would lead to the belief that its use might increase the incidence. Garnett Wright,⁵⁴ in his series of 155 cases, found 13 in which portions of such suture could be found. Many other similar cases have been reported, but Huist²⁴ believes that the importance of this factor has been exaggerated, as pieces of suture may be present without ulceration. In my own 79 cases there were 15 examples, in 2 of which I had performed the primary operation myself. In some of these the suture arose from the centre of the ulcerated area and it was difficult not to conclude that it was a factor of some importance (*see Fig 19*). But since in 64 cases no suture could be found even by microscopical examination, it is evident that this is not so important a factor as was at one time believed. The use of catgut may reduce the incidence but it will not abolish it.

It has been claimed that the crushing effect of clamps applied during the process of anastomosis might initiate the ulcer, which would slowly progress. The main answer to this contention is that, as with ulcers of the stomach and duodenum, it is now known to be a very easy matter to produce acute ulceration experimentally, but unless some other factor is present, such ulcers always heal rapidly and cannot be induced to progress to a chronic state. If the other factor is present, ulceration will occur with or without the presence of primary trauma. Bolton⁷ states that an acute toxic ulcer produced in an animal invariably heals soundly in three to four weeks. Paterson⁴⁰ states that there are two main reasons against the acceptance of this view—firstly, that there is too long an interval between the operation and the onset of symptoms of marginal ulceration, and, secondly, that the obstruction does not as a rule occur at the site where the intestine is gripped by the clamps. In the 79 cases in this series there was not one in which the ulcer was so situated as to give rise to the belief that it could have originated in this way. Gronnerud²⁰ carried out a series of experiments in dogs showing that the incidence of this complication was unaffected by the use of clamps, and in many of the reported cases in man no clamps have been used. It may therefore be accepted that if clamps are employed with ordinary care they are not a factor in the production of this lesion.

It would seem reasonable to believe that if care were not used to obtain accurate apposition of the mucosa a raw area might be left at the line of suture which, after being acted upon by the acid juice, would progress to a chronic peptic ulcer. Grey Turner¹⁹ has laid stress upon this possibility and shown the importance of obtaining accurate mucosal apposition. My attention was first directed to this possibility by the fact that several of the ulcers in those cases where I performed the first operation

myself were placed in a different situation from those seen when the first operation had been performed elsewhere. The majority of surgeons terminate their anterior line of through-and-through sutures at one or other extremity of the mucosal incision and it was in this position the new ulcer was found (*see Fig 17*). It has been my own custom to terminate this suture about the middle of the incision so that I turn the corners with a continuous suture. In several of my cases the ulcer was situated at this point (*see Fig 18*). In either case the ulcer was placed at the last half inch of the through-and-through suture line where the mucosa is most likely to slip back and thus escape inclusion in the union. For this reason I have for several years past abandoned excision of the redundant mucosa. The line of suture is less neat in appearance, but one has the feeling of certainty that there is complete mucosal apposition throughout the whole length. Even with such precautions ulceration still takes place. If the formation of a raw area is a factor, it is a small one.

At one time great stress was laid upon the effect of pyloric occlusion in increasing the incidence of gastrojejunal ulceration. Martin³³ supports this view, for he states that the pancreatic secretion is normally activated by the passage of acid through the pylorus. The contact of this acid with the duodenal mucosa produces secretin, which is carried by the blood-stream to the pancreas, where it stimulates the secretion of pancreatic juice. If the pylorus is occluded, this mechanism is disturbed. Galpern¹⁶ stresses the importance of this fact in surgery and shows that if a dog's stomach is divided into two halves, the introduction of acid into the cardiac end has no effect, but if it is inserted into the pyloric portion a flow of pancreatic juice immediately follows. Von Haberer¹⁷ supported this view on his clinical experience, for he found that after 275 cases of simple gastro-enterostomy there were 3 examples of jejunal ulcers, and after 71 cases with pyloric occlusion there were 12 jejunal ulcers. Paterson⁴¹ is, however, entirely opposed to this view, for he has found that after gastro-enterostomy there is a diminution of the total chlorides as well as of the free HCl in the gastric juice. He regards this as being due to the fact that the hormone regulating the flow of HCl is formed in the pyloric end of the stomach and is not utilized if the food does not enter this portion. Whatever is the effect of attempted permanent occlusion is of minor importance, however, as this step is rarely practised to-day. Temporary occlusion with a silk stitch would seem to have very little bearing. I invariably practice this step, but the incidence of marginal ulceration in my own cases has been no higher than in series reported elsewhere.

As a result of this tedious but necessary discussion it would appear that the following conclusions can be accepted as accurate: (1) Marginal ulceration is almost limited to cases of anastomosis for duodenal ulcer, (2) After all gastric operations its incidence is 1.6 per cent, (3) After all gastric operations watched for ten years its incidence is 2.3 per cent, (4) After all posterior gastro-enterostomies its incidence is 2.2 per cent, (5) After all posterior gastro-enterostomies for duodenal ulcer its incidence is 3.24 per cent, (6) After all posterior gastro-enterostomies for duodenal ulcer watched for ten years its incidence is 3.9 per cent (this must be considered as the highest possible incidence), (7) The condition is less common after partial gastrectomy, the incidence being only 0.61 per cent, (8) Technical errors in the primary operation may increase the frequency of the lesion, but are not the primary cause.

THE CHOICE OF OPERATION

Since after partial gastrectomy there is a much lower incidence of marginal ulceration, it would seem evident at first sight that it should be the operation of choice, but it is necessary to be certain before a decision is made that this more extensive operation is not associated with a higher mortality. In investigating such a question there are always great difficulties, for the operative mortality is the one fact which alters with the experience of the surgeon and therefore is to a large extent personal. Much time is wasted in surgical meetings discussing the best operation for a given lesion. Preferably the question should be: Which is the best operation in the hands of a given surgeon? One surgeon may perfect one method and another a second. Nevertheless these operations have now been so widely performed that a fair estimate can be made of the operative mortality with either method obtained by the more experienced surgeons.

Luff,³⁰ in his collective investigation, gave the total mortality for posterior gastro-enterostomy as 5 per cent, and Conybeare¹² states that of 190 cases admitted to Guy's Hospital the mortality was over 5 per cent. Individual series show, however, much better results. Mayo³⁵ obtained in 4532 cases of duodenal ulcer a mortality of 1.76 per cent. Balfour³ found in 492 cases 9 deaths—that is, 1.82 per cent—but in his report for 1930² there were 524 cases with only 6 deaths—that is, 1.14 per cent. Lord Moynihan's series of 1000 cases without a death has frequently been commented on. These are, however, the statistics of exceptionally skilled surgeons, and can be equalled by but few. In this country, indeed, there is a tendency for the mortality of gastro-enterostomy in the hands of specialists to increase, for the simple cases are now performed locally and only the patients who—owing to old age, concomitant disease, past severe hæmorrhage, or extreme stoutness—must be considered as bad surgical risks are sent to the specialists. In my own series analysed up to November, 1929, there were 550 cases with 7 deaths—that is, a mortality of 1.2 per cent—and this included a consecutive series of 263 cases without a death. In the series analysed up to August, 1933, the total mortality was 2 per cent, and it is probable that this figure should be regarded as the average mortality for this operation. Although perhaps not germane to the present discussion, it must also be remembered that many surgeons have been able to publish series of gastro-enterostomies giving 80 to 90 per cent of cures. I have published⁵² a series of my own cases where 88 per cent were cured after being followed for a minimum of five years.

The mortality of partial gastrectomy performed for duodenal ulcer is also difficult to estimate, for many of the reported series of partial gastrectomies include cases of ulcer of the body of the stomach, so that the two series are not comparable. Probably the most satisfactory figures that have yet been published are those of Finsterer,¹⁴ who in a total of 566 cases had only 18 deaths, a mortality of 3.1 per cent. But with the Billroth I method it was 5.8 per cent. Burke¹¹ gives the results of cases operated upon at the Eiselsberg Clinic in Vienna, there were 606 cases with a mortality of 5.44 per cent. Louria²⁹ reports the results of cases operated upon by von Haberer at the University Clinic at Graz, there were 284 cases with 24 deaths, a mortality of 8.4 per cent. Schoemaker⁴³ reports results of 350 cases operated upon by his method, there were 17 deaths—that is, 5 per cent. Balfour in 1929 published a series with a mortality of 5.5 per cent, and the collected statistics

of Luff³⁰ gave a 6 per cent mortality. In my own series a partial gastrectomy was so seldom performed for a duodenal ulcer that I have no comparable statistics.

From the above figures it would seem fair to estimate that the mortality after gastro-enterostomy for duodenal ulcer is in the neighbourhood of 2 per cent and after partial gastrectomy about 6 per cent. That is, the mortality of the latter operation is slightly higher than the combined mortality and the incidence of recurring ulcer after posterior gastro-enterostomy, and this does not take into consideration the incidence of recurring ulcer after partial gastrectomy.

Since, as will be shown later, at least 75 per cent of gastrojejunal ulcers can be cured by a subsequent partial gastrectomy it would seem manifest that the most satisfactory operation for duodenal ulceration is posterior gastro-enterostomy, a partial gastrectomy being reserved for the small percentage of cases which later develop gastrojejunal ulceration.

PATHOLOGY

The pathological characters of these ulcers are identical with peptic ulcers seen in the stomach or duodenum. A few cases of acute ulceration are seen, these are either operated upon for severe hæmatemesis or are discovered post mortem after a perforation or death from some other cause, they are therefore usually found shortly after operation. In 3 of the 79 examples in my series there were intervals of one month, one month, and five months respectively after the first operation. In such cases there are one or more areas of sharply cut but irregular ulceration involving only the mucosa but tending to encircle the anastomotic area. The edge of the mucosa may be free or only lightly attached to the underlying tissue and there is little or no induration around. The surface may be covered with sloughing material, and small areas of hæmorrhage or a small vessel which is eroded may be visible. Rarely in these early stages an acute ulceration may progress to perforation, in which case a relatively acute necrosis with but slight reactionary induration is seen. Such examples appear to occur most commonly in the course of some acute infectious complication. It is probable that nearly all anastomoses would show a certain amount of superficial ulceration shortly after operation owing to necrosis from pressure of the continuous suture, but only a few progress to the chronic stage.

Long-standing ulcers have all the characters of the chronic peptic ulcer, being deeply cut and penetrating. They tend to be circular, the mucosa is turned in and attached to the edge of the destroyed muscle, the floor is formed either of thickened peritoneum or some adherent neighbouring structure, and if sections of it are cut there is a complete absence of muscle fibre. There is much infiltration and fibrosis, at first of the surrounding wall of the stomach and jejunum, and later of adherent tissues such as the mesocolon, colon, or anterior abdominal wall. Like chronic peptic ulcers of the stomach there is usually a considerable increase of the fat of the surrounding peritoneum. They are usually single, and may be localized or extend nearly around the anastomosis. If single they are most commonly at the extremities where the afferent or efferent loop is united to the stomach (*Fig 17*), but in some of the cases where I had performed the primary operation they were situated at the middle of the anastomosis (*Fig 18*). The possible reason for this site has been considered in the discussion on operative technique. In some cases they are

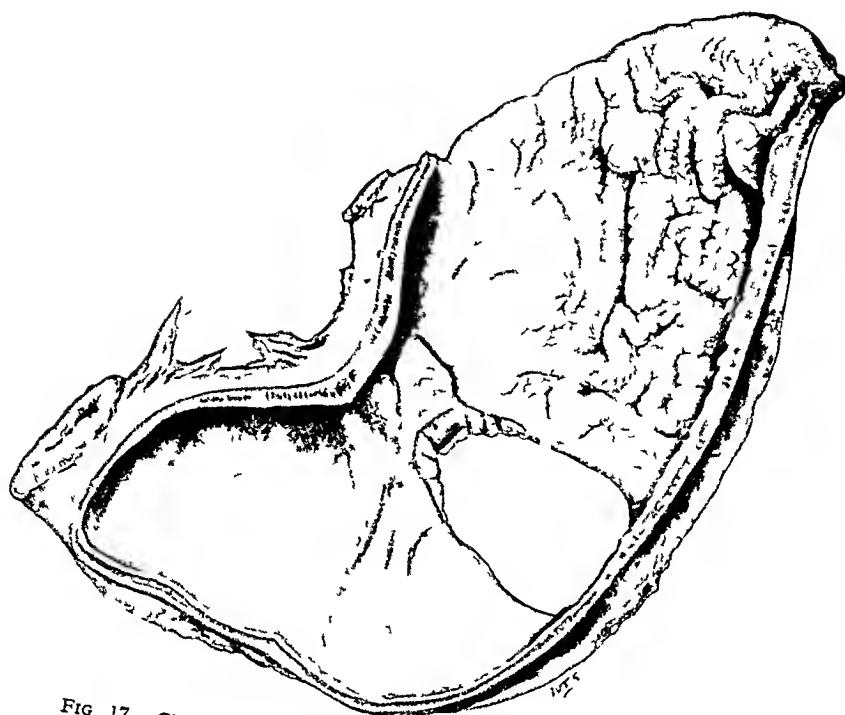


FIG 17—Chronic gastroduodenal ulcer at upper margin of stoma

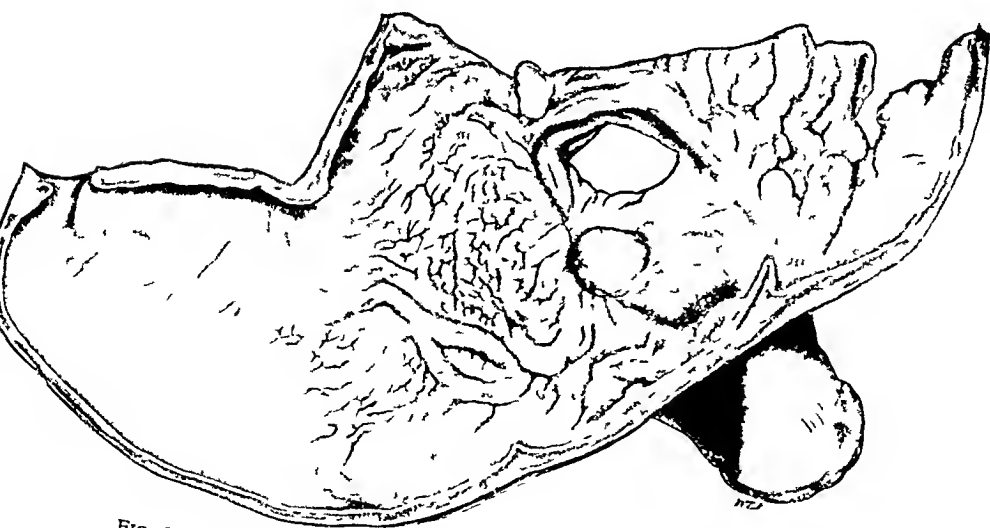


FIG 18—Chronic gastroduodenal ulcer at centre of margin of stoma

multiple, as many as three or four having been reported Paterson³⁹ found that in 52 of his cases in which the nature of the ulcer was described, 6 were multiple

In some cases pieces of unabsorbed suture are clearly visible passing from the floor of the ulcer (*Fig 19*), and in others small portions of such sutures are found only on close examination In many, however, the most careful search with the naked eye and microscope fails to reveal their presence

Paterson³⁹ laid great stress upon the distinction between gastrojejunal and jejunal ulcers, and this distinction has generally been maintained He stated that the gastrojejunal ulcers involved the gastric mucosa, whereas the jejunal were found remote from the anastomosis He also believed that the characters of the two ulcers differed, those in the jejunum, both in appearance and clinical course, bear

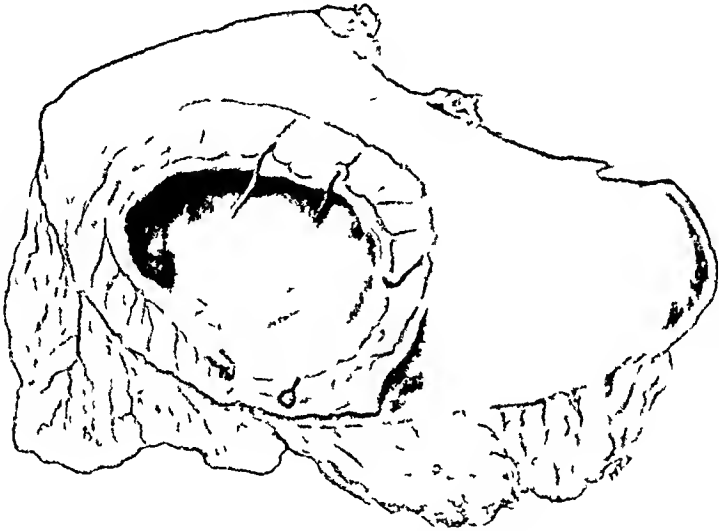


FIG 19 —Gastrojejunal ulceration around portions of silk suture

a close resemblance to the ordinary round ulcer of the stomach and duodenum, and they are apt to perforate before protective adhesions are formed They are most common in the efferent limb, either opposite or just below the anastomosis He states that the gastrojejunal ulcers lack the definite localized appearance of the jejunal ulcers and occur as an irregular ulceration around the margin of the anastomotic opening After healing occurs the process of cicatrization may lead to partial or even complete stenosis of the anastomotic opening It is probable that such stenosis is always due to an old marginal ulcer These ulcers are less likely than jejunal ulcers to perforate into the general peritoneal cavity Other observers have accepted these views, and cases have been recorded where the ulcer in the jejunum was situated several centimetres away from the margin In my own series of 79 cases it was not found possible to make this distinction Some of the ulcers which involved a large portion of the margin spread widely into the stomach, or extended well on to the jejunum Even in those cases in which the ulcer appeared to be separated from the line of junction there was always an area of scarred tissue spreading from this line to the actual ulcer (*Figs 20, 22*) In fact in no single case in the series was it possible to say definitely that the ulcer was jejunal, and therefore

the evidence at my disposal at present would lead to the belief that all such cases start at the anastomotic union

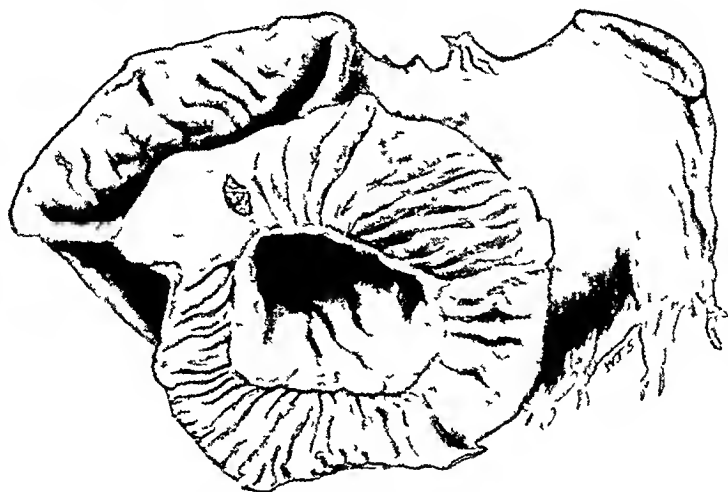


FIG 20 —Ulcer, apparently jejunal, connected by scar to margin of stoma

As in the case of chronic ulcers of the stomach and duodenum the ulcer in its progress tends to cause inflammatory changes in surrounding tissues, which become thickened and infiltrated. In the case of a posterior gastro-enterostomy the involved

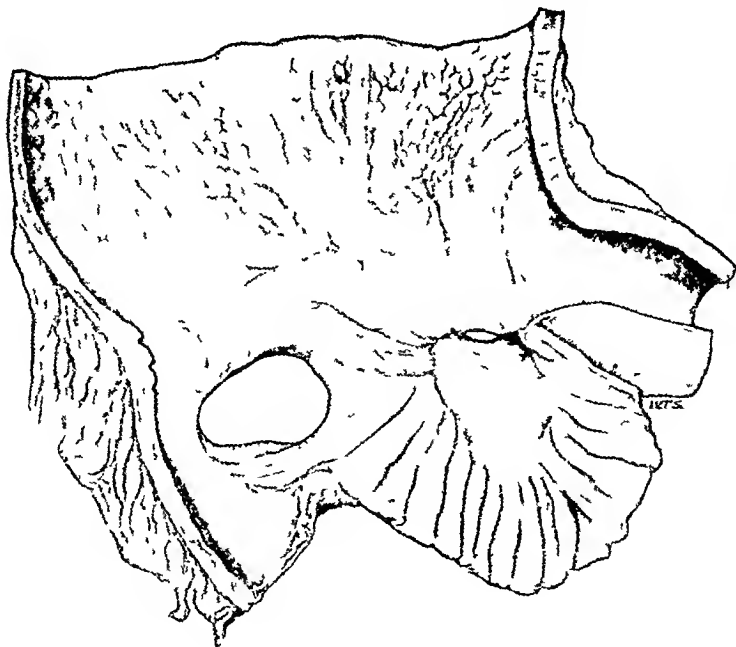


FIG 21 —Chronic ulcer with perforation at margin of anterior gastro-enterostomy stoma

tissue is the mesocolon, and as this becomes infiltrated it is shortened until the transverse colon is bound down and becomes firstly adherent to the base of the ulcer and then perforated, so that a gastrojejuno-colic fistula forms and the food may

pass directly from the stomach to the colon. Such fistulae are generally only found in males, as were all four cases in my series.

In the case of an anterior gastro-enterostomy (*Fig 21*) or of a Roux operation (*Fig 22*) there is not the same close proximity of the mesocolon, and the base of the spreading ulcer may become adherent to the anterior abdominal wall. It may then give rise to a tender mass, or an abscess may form in the substance of the abdominal muscles. In some cases, indeed, as in one of Paterson's, the abscess may break down and an external fistula form. If the ulcerating process is rapid and acute, or if no adhesions have been formed to the surrounding viscera or to the abdominal wall, perforation into the peritoneal cavity may occur in precisely the same way as a gastric or duodenal ulcer may perforate. It is of interest to note that in the earlier published series nearly all the cases had perforated into the peritoneum or into some surrounding tissue. Thus Paterson in 1909 classifies his 52 certain cases into (1) Perforations into the peritoneal cavity, 19 cases, (2) Non-perforations, 33 cases. These latter he again divides into two classes (a) penetrating abdominal wall, 28 cases, (b) penetrating some hollow viscus, e.g., the colon, 5 cases.

To-day the condition is so well recognized and the symptoms are so well known that the ulceration is as a rule diagnosed long before either perforation or penetration into a surrounding viscus has occurred. Thus in Garnett Wright's series⁵⁴ published in 1920 there were only 31 perforations in a total of 155 cases. It is interesting to note, however, that Bolton⁸ in the same year recorded 4 cases of jejuno-colic fistula and stated that his cases brought the recorded total of such up to 31. In my own series of 79 cases the majority were diagnosed on the symptoms of the uncomplicated ulcer, and there were in all only 6 perforations and 4 colonic fistulae. Perforation to-day is indeed only likely to be found in the acute cases, and may occur within a few months of the performance of the gastro-enterostomy.

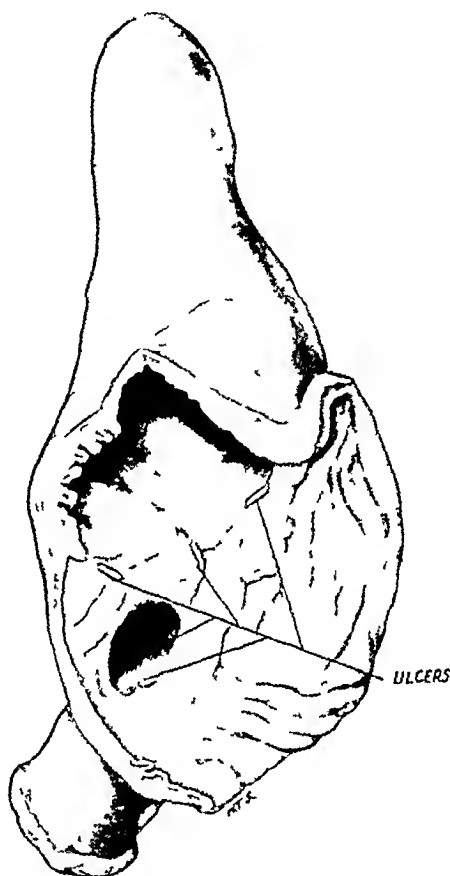


FIG. 22.—Multiple ulcers round stoma of Roux or in Y gastro-enterostomy.

ETIOLOGY

It has long been recognized that these ulcers are much more common in males, and this sex distribution seems to be more evident in the more recent series, possibly because the contributory factor of errors in operative technique is being eliminated. In Paterson's series the sex was mentioned in 50 cases, 39 being men and 11 women. Hurst states that there are about 6 males to 1 female. In my 79 cases

there were only 7 females, and in the 30 in whom I had performed the first operation only 1 was a female. There is often a family history. This is a part of the family history of primary duodenal ulcer with which all surgeons are acquainted, for Huddy²³ found in 129 cases of duodenal ulcer that 56 (42 per cent) showed a family history. Hurst, however, has shown that this inherited tendency goes deeper than the actual ulceration and that it is the variety of gastric function which runs in the family, a view which is supported by Apperly and Norris,¹ who investigated carefully a series of families comprising 86 individuals. In confirmation of this view, Hurst has been able to show that there is actually a family history of the gastrojejunol ulceration, he was able to obtain positive evidence of this in 6 out of 43 cases seen at the New Lodge Clinic, an observation which becomes of extreme interest when the cause of the marginal ulceration is considered.

As would be expected, since most of these cases follow operations for duodenal ulcer, they are generally men of middle age. Paterson's series, however, contains an interesting example in a female aged 2 months, the gastro-enterostomy having been performed for congenital pyloric stenosis.

The important questions that arise, therefore, are: Why should gastrojejunol ulceration occur practically only after the operation has been performed for duodenal ulcer? And why is it so much more common in males? Primary peptic ulcers are almost unknown in the jejunum. Occasionally one is reported which appears to be peptic in nature and is not dependent upon any specific infection. I have described an example⁵⁰ occurring in my own practice, and Richardson,⁴² in reviewing the literature of this subject, reported 2 cases of his own where ulcers, apparently peptic in type, occurred 2 ft. from the flexure and about the middle of the jejunum respectively. He was able to find records of 10 other published cases. Of the total of 12, 10 occurred in men. Nevertheless these examples must be regarded almost as pathological curiosities.

Stress has been from time to time laid upon the importance of infection as a factor leading to the development of marginal ulceration. Hurst and Paterson have both discussed this point and my own experience would agree with theirs, that infection anywhere in the body is likely to increase the incidence, especially of the acute ulcers following shortly after operation. It is, however, not the sole cause, for infection is not more common in men than in women, and is not likely to be more frequent after anastomosis for duodenal ulcer than for any other lesion.

The important factor has now definitely been proved both by experiment and clinical observation to be the presence of hyperchlorhydria. Montgomery³⁶ found that gastrojejunol ulceration developed after gastro-enterostomy in dogs just as it may do in men. In a series of 60 such operations this form of ulceration was found in 4. Mann and Williamson,³² acting on the assumption that the acidity was the cause of peptic ulceration, divided the pylorus, closed the proximal end of the duodenum, divided the ileum, joined the distal end of this to the pylorus, and implanted the proximal end into the ileum lower down. By this means the alkaline bile and pancreatic juices were transferred to the intestine below the anastomosis. A large number of the animals developed typical peptic ulcers. The whole of the experimental work has recently been carefully reviewed and further experimental proof provided by Matthews and Dragstedt.³⁴ They formed gastric pouches which were joined to the distal end of the divided intestine, the proximal end of the intestine being implanted into the ileum well below the junction of the gastric

pouch In 6 cases where the gastric pouch was united to the ileum all developed peptic ulcers In 13 where it was united with the jejunum 11 developed ulcers Similar ulcers developed in gastric transplants made into the intestine low down Such transplants secrete their own acid, the secretion, as shown by Ivy and Farrell,²⁵ being stimulated by gastrin bodies carried by the blood-stream The acid acts on the mucosa long before the food could come down the intestines to neutralize it Transplants of intestine into the stomach wall, on the other hand, never develop ulcers The removal of the saliva by the formation of an œsophageal fistula or excision of the salivary glands was not followed by ulceration, which agrees with the clinical observation that peptic ulcers do not occur in cases of gastrostomy performed for complete œsophageal obstruction They prove by a series of ingenious experiments that the important factor is the removal of the alkaline duodenal fluids, and of these the pancreatic secretion is the more important When the bile was led into the stomach but the pancreatic juices were removed all the animals but one developed peptic ulcers—an interesting commentary on the operation of cholecystogastrostomy, which has been advocated by some as a cure for gastric and duodenal ulcers Bollman,⁶ however, found that after experimental obstruction of the common duct there were 64 examples of peptic ulceration and only 23 without ulcers The earliest ulcer was found 5 days after operation and the latest 295 days They appeared most commonly 70 to 90 days afterwards

Somewhat similar conditions may be found in man It is well recognized that peptic ulcer is sometimes found in a Meckel's diverticulum and is then dependent upon the presence of heterotopic areas of gastric mucosa, this condition corresponding exactly with the experimental transplants of gastric mucosa In some cases an operation carried out for the cure of a definite lesion may, just as in the experiments, remove the juices which neutralize the acid and then be followed by peptic ulceration I have personally reported⁵¹ two such cases In the one another surgeon in performing a partial gastrectomy for what was believed to be carcinoma of the stomach excised the body of the pancreas The lesion was proved to be a simple ulcer, but the removal of so much of the pancreatic juice was followed by the formation of a large gastrojejunal ulcer A more extensive partial gastrectomy, by further lowering the acidity, was followed by a cure In the second case a cholecysto-enterostomy was performed for obstructive jaundice due to chronic pancreatitis This was followed some years later by the development of a duodenal ulcer If a lateral anastomosis is carried out in addition to the gastro-enterostomy or an anastomosis 'in Y' is performed, a condition is produced very similar to that in the experiments of Mann and Williamson, in that the alkaline secretions are made to enter the intestine below the anastomosis, and hence peptic ulceration would be expected to follow

On the clinical side the importance of hyperacidity has also long been recognized Paterson in 1909 investigated the acidity of the gastric juices obtained from the fistula in one of his cases and found definite hyperacidity He discussed fully the evidence in favour of such acidity being the cause of the ulceration It is to Hurst, however, that we owe the chief work on this subject He shows that the frequency of marginal ulceration varies directly with the hydrochloric acid content of the gastric juice, and produces strong evidence in support of this contention It is for this reason that marginal ulceration is almost unknown in carcinoma and is so rare in cases of ulceration of the body of the stomach, whereas with duodenal

ulcer, where the acidity is high, ulceration is more common. He found that when the gastro-enterostomy is undone for jejunal ulceration there is an exceptionally high curve of acidity, indicating that an extreme degree of the hypersthenic gastric diathesis may be a predisposing cause. The presence of such hyperacidity would also account for the familial occurrence. My own experience is in accord with this evidence. I was able to obtain the results of the test-meals at the time of the first operation in 28 of the 30 cases where I had performed this operation myself, and in 4 of the 49 where the operation had been performed elsewhere. The average figures in these 32 cases were free HCl 0.203 and total acid 73. In my own cases the average of 21 cases at the time when the marginal ulcer was found was 0.14 and 54, and in 28 of the remaining cases 0.13 and 51—figures showing a very marked hyperchlorhydria.

Of even greater interest was an investigation which I have previously published,⁴⁸ the figures of which (*Table VI*) show that the average acidity was always higher with duodenal ulcers than with lesser-curve ulcers. It was always higher in males than in females, and, what was of exceptional interest, it was highest in those cases of duodenal ulcer which later developed marginal ulceration. At that time I came to the conclusion that the development of gastrojejunal ulceration was probably due to an anatomical variation occurring in 3 to 4 per cent of men, wherein there was a wider distribution than normal of the acid-secreting cells of the stomach—a theory not very remote from Hurst's belief that there is in these people an extreme degree of the hypersthenic gastric diathesis.

Table VI—AVERAGE TEST-MEALS

	NO OF CASES	AVERAGE FREE HCl	AVERAGE TOTAL ACID
Lesser curve (females)	57	0.108	51.0
Lesser curve (males)	135	0.123	61.0
Duodenal (females)	44	0.136	57.6
Duodenal (males)	213	0.165	63.5
Duodenal, later gastro- jejunal	13	0.190	71.0

On these considerations it seems permissible to accept the following (1) Gastrojejunal ulceration is only common after gastro-enterostomy for duodenal ulcer, (2) It is much more frequent in males, (3) It occurs after 3 to 4 per cent of all gastro-enterostomies for duodenal ulcer, (4) It is directly due to hyperacidity, (5) The hyperacidity is a congenital and possibly a familial fault.

SYMPTOMS

It has already been mentioned that in the earlier published series the condition was usually not diagnosed until perforation or penetration had occurred. In the more recent series these complications are much rarer, for the symptoms of non-penetrating ulcers are now well recognized and the condition treated. It is probably because of these earlier series that the statement is still so often made that there is frequently a long latent period, perhaps of many years, before symptoms arise. Such a history is indeed often obtained to-day in a patient who is first seen

when the ulcer is well established. Although such patients will state that for years after the operation they are quite well, the one fact that has been impressed upon me by watching them carefully in a Follow-up Department is that there is generally no latent period. A patient with a gastro-enterostomy for a duodenal ulcer is, as a rule, one of the most satisfactory of our cases. He is soon able to eat all food with no thought or care, and is able to carry out his normal work. His usual remark is that he has never had a day's discomfort since the date of his operation, and his presence in the Follow-up Department does much to encourage the surgeon in his many disappointments. The patient who later develops a marginal ulcer does not as a rule make this satisfactory progress. He is well, but has a little flatulence or he has to be careful of his food, he occasionally has nausea. He cannot undertake heavy work or he has to continue with his bismuth and alkali long after the usual period, or symptoms will return. I have learnt now to regard all such cases with considerable mistrust, especially if they had an unduly high acidity before the operation. They will generally develop characteristic symptoms of marginal ulceration, which may not appear for some years, at which time they will have forgotten their earlier minor symptoms. In the 30 cases of my own series the average time elapsing between the gastro-enterostomy and the onset of definite symptoms was 2 years and 11 months, but in two cases it was only a month, and in one was as long as 14 years.

Clinically there are two definite groups. In the first the dominant symptom is hæmorrhage. This may commence as a mild hæmatemesis or melæna shortly after operation, or the patient may continue in a very fair state of health for a year or more and then have a sudden severe loss of blood, generally in the form of melæna. In the earlier cases this symptom was often regarded as evidence of persistence of the duodenal ulcer, but, as I have frequently stated, I have never yet seen a duodenal ulcer persist after an adequately performed posterior gastro-enterostomy. I believe that all such cases have marginal ulceration, but as at this stage it may be only acute, it may be readily overlooked at operation or even indeed at a post-mortem examination. In my series of 79, 32 had had hæmorrhage, but in many of these it occurred later when other symptoms were present.

In the more common variety there is in the earlier stages only a minor degree of discomfort, fullness, or flatulence, which is later replaced by a definite pain, at first thought to be that of the old duodenal ulcer. It simulates the original lesion in that it becomes periodic, so that the patient may have severe attacks lasting for two or three weeks and may then be quite free for several months. It is severe, occurs late after food, is relieved by food, and often wakes him at night. A careful investigation will show, however, that the pain differs from that of a duodenal ulcer in that it is often referred to the lower abdomen, and especially to the left iliac fossa, and that vomiting, which is so rare a symptom with uncomplicated duodenal ulcer, is often present and generally gives transient relief to the pain. In the latter stages, as the ulcer penetrates, the pain becomes more and more severe and much more constant. It may indeed become agonizing and the patient pass into a stage of deplorable misery. In my 79 cases pain was present in 73, and it must therefore be regarded as the most frequent and characteristic symptom. Vomiting, generally giving temporary relief, was present in 46 cases. In the earlier stages the patient retains his appetite and the test-meal shows a high acidity. Later when the pain becomes more severe his appetite may begin to fail.

The value of X-ray investigation has been much disputed. There is no doubt that in the earlier stages very little can be seen, and even in the later stages considerable skill is required to interpret the findings. The presence of tenderness over the visualized stoma is a finding of considerable value, as are hyperactivity and rapid emptying. Irregularities in the margin of the stoma and above all the presence of a well-marked meniscus close to the stoma confirm the diagnosis (*Fig 23*). In my own series the very skilful work of Dr Vilvandre has been of great help, and he was able to make a positive diagnosis in a high percentage of the cases.

Of the symptoms of perforation nothing need be said, for they differ in no way from the symptoms of a similar complication of gastric or duodenal ulcer.

When penetration has occurred and a fistula passes into the colon the clinical picture may be considerably altered, the cases generally falling into one of two groups. In the one they are dependent upon the presence of a free opening between the stomach and colon. According to Bolton, if the ulcer had been gastro-jejunal, the opening between the two viscera is direct and the dominant symptom is faecal vomiting and the eructation of foul gas. If, however, the ulcer had been jejunal, the communication will be indirect through the jejunum, in which case the chief symptom is diarrhoea, due to irritation of the colon from the presence

of the gastric contents. In practice it is, however, difficult to make this distinction. It is true that in some cases the presence of colonic content in the stomach with foul vomiting, eructation, loss of appetite, and wasting are the chief symptoms, but even then attacks of diarrhoea are frequent and the patient feels his best when constipated. If the colonic symptoms are more marked, there will generally be found some evidence of gastric regurgitation and gastritis. In either case the examination of the test-meal will reveal the presence of faecal material, but the certain diagnosis to-day rests upon the X-ray examination. The barium meal will be found to enter the colon almost at once, or a barium enema will pass through into the stomach. In such patients the pain is very varied and often the severity seen with the original ulcer seems to be diminished, probably because of the neutralization of the acid by the colonic contents or its diminution owing to the resulting gastritis. Hurst has made the observation that with the formation of a fistula the ulcer usually heals. In some cases, however, the pain remains severe. The patients are usually wasted and often indeed emaciated.

In the second group the dominant symptom is one of intestinal obstruction, for it will nearly always be found that the fistula is associated with much fibrosis, which will spread around the colon and thus narrow its lumen. In these patients

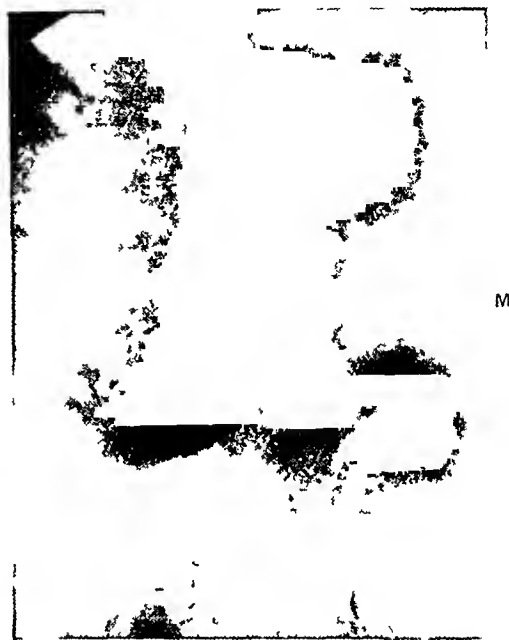


FIG 23—X-ray showing meniscus (M) of large gastrojejunal ulcer

there will be attacks of colicky pain with dilatation and visible peristalsis of the proximal colon, but even in this group attacks of faecal vomiting may occur, and the patients are nearly always very wasted. The X-ray investigation, as in the last group, will generally reveal the presence of a fistula as well as the obstruction.

When the anastomosis has been anterior the penetrating ulcer usually becomes adherent to the anterior abdominal wall, generally behind the left upper rectus. Rigidity and tenderness of this muscle may be seen in the earlier stages, and later a swelling develops in the substance of the muscle. If left untreated, this becomes red and oedematous, and later breaks down so that a gastric fistula is formed through which much of the gastric contents may escape and lead to digestion of the skin. Like the colonic fistula, these are becoming rare to-day, as treatment is usually undertaken before their inception.

TREATMENT

As with the original peptic ulcer, there is considerable divergence of opinion between physicians and surgeons concerning the value of medical treatment. Hurst²⁴ states that with careful medical measures gastrojejunal ulcers can be cured, but he also very fairly remarks that if the regulations are disobeyed the ulcer tends to recur, and that if severe obstruction is present or prolonged medical treatment fails, resort should be had to surgery. Of 25 of his cases treated medically, 13 were more or less completely relieved. My own experience of medical treatment has, however, been very unsatisfactory. The aid of the physician has always been sought in the earlier stages of this condition occurring in my own practice, but wherever the diagnosis could be considered as certain the lesion has always progressed and surgery has later been necessary. In every other case where the first operation had been performed elsewhere medical treatment has been tried before my interference had been sought, but it is only fair to state that of necessity it is only the failures which are sent to me, and I have therefore no means of knowing how many may have been cured.

If surgical treatment is required, Hurst believes that the best as well as the simplest operation is local excision. Such is not my experience. In my earlier cases, acting on the belief that these ulcers were generally dependent upon the use of unabsorbent sutures, a local excision was performed and the opening re-sutured with catgut. There were 13 such cases, of which 1 died after operation. Of the remaining 12, 6 recurred and further operation was required. It is true that some of these patients, and especially those with a colic fistula, are extremely ill and will be able to stand only the simplest operation. In these cases separation of the fistula with local excision of any ulcer and closure of the openings may alone be possible, but such steps should always be regarded merely as temporary measures to save the life of the patient. He must be carefully treated and watched afterwards, and, if the ulcer persists, the more complete operation performed when his condition has improved.

The operation which at one time was very popular and still has its advocates is excision of the ulcer, separation of the anastomosis, and closure of the two openings, thus restoring the condition to normal. In some cases there is so much stenosis of the duodenum from healing of the old ulcer that such a step is only possible if a gastroduodenostomy or some similar step is performed at the time

This operation is, however, theoretically unsound, as it restores the tendency for the formation of a duodenal ulcer and there is no reason to believe that after the passing of time the hyperchlorhydria is diminished. Hurst lays stress upon this point and states that if the opening is closed a fresh ulcer may develop. There were 2 examples in his series of 44 cases. One case of such recurrence after closure of the opening by another surgeon occurred in my series, but I have no statistics of its frequency, for I have never performed the operation.

The method which would seem to offer the most satisfactory results is that of partial gastrectomy, for it has been shown that this operation is followed by a much lower incidence of marginal ulceration than is gastro-enterostomy. It is, however, an operation of very considerable severity. Much time may be occupied in freeing adhesions and making the anatomical relationships manifest before the actual resection is commenced. The operation becomes in fact one of the most exacting and difficult procedures within the abdomen. Being performed, as it is so frequently, on patients who are often exhausted by long-continued severe pain, by vomiting, by hæmorrhage, or by malnutrition from the presence of a fistula, it is likely to be associated with a high mortality. For this reason Hurst condemns it and states that it is only advisable in very exceptional conditions. He quotes von Eiselsberg⁴⁵ as having a mortality of 25 per cent for such operations. He also quotes Klein²⁷ as finding that only 2 out of 6 of these operations were followed by achlorhydria, and therefore suggests that the risk of a new ulcer remains in 66 per cent. There is no doubt that in the earlier operations performed by all surgeons the mortality is high, but as experience is gained in the selection of cases for operation and the performance of temporary palliative operations upon those who are dangerously ill, this can be greatly reduced. Finsterer¹⁵ had a mortality of 9 per cent in 76 cases. In my own series of 79 cases a partial gastrectomy was performed in 61, and 16 of these patients died, a mortality of nearly 25 per cent, but most of these deaths occurred in the earlier cases before experience was gained, and in the last series of 20 cases there was only 1 death, a mortality of 5 per cent. The risk of recurrence after this operation is very slight. Finsterer states that if three-quarters of the stomach is removed recurrence never takes place. In my own series of 61 cases there has only been 1 recurrence, this being the one already described, where owing to adhesions too small a portion of the stomach was removed. A later and larger resection was followed by a complete cure.

It would therefore seem reasonable to accept the view that partial gastrectomy, although not the operation of choice for duodenal ulceration, is so for gastrojejunal ulceration, and should only be replaced by more simple methods if the condition of the patient will not warrant its use.

REFERENCES

- ¹ APPERLY, F. L., and NORRIS, J. H., "The Familial Influence in Gastric Function", *Brit. Med. Jour.*, 1931, 1, 255.
- ² BALFOUR, D. C., "Annual Report of Surgery of the Stomach and Duodenum for 1930", *Proc. Staff Meetings Mayo Clinic*, 1931, Jan. 28, 55.
- ³ BALFOUR, D. C., "Report on the Surgery of the Stomach and Duodenum in the Mayo Clinic for 1929", *Ibid.*, 1930, March 5, 64.
- ⁴ BEER, I., "Contribution to the Etiology of Peptic Ulcer of the Jejunum", *Zentralbl. f. Chir.*, 1922, 21, 282.

- ⁵ BIRGFELD, E, *Arch f klin Chir*, 1925, cxxvii, 568
- ⁶ BOLLMAN, J L, "Peptic Ulcer in Experimental Obstructive Jaundice", *Proc Staff Meetings Mayo Clinic*, 1930, Dec 10, 357
- ⁷ BOLTON, C, "Discussion on the Prognosis of Peptic Ulcers", *Proc Roy Soc Med*, 1934, Jan, 226
- ⁸ BOLTON, C, "Discussion on the Remote Results of the Surgical Treatment of Gastric and Duodenal Ulcers", *Ibid*, 1920, May, 166
- ⁹ BRAUN, *Verhandl d deutsch Gesellsch f Chir*, 1899, ii, 94
- ¹⁰ BRODNITZ, *Ibid*, 1903, i, 77
- ¹¹ BURKE, J, "The Operative Mortality and Morbidity of Partial Gastrectomy for Peptic Ulcer", *Surg Gynecol and Obst*, 1931, Nov, 704
- ¹² CONYBEARE, J, "Discussion on the Prognosis of Peptic Ulcers", *Proc Roy Soc Med*, 1934, Jan, 228
- ¹³ EINER KAY, *Bidrag till den Kirurgiska Behandlingen af Ulcus Vetriculi*, 1907 Stockholm
- ¹⁴ FINSTERER, H, and CUNHA, F, "The Surgical Treatment of Duodenal Ulcer", *Surg Gynecol and Obst*, 1931, June, 1099
- ¹⁵ FINSTERER, H, *Brit Med Jour*, 1926, ii, 553
- ¹⁶ GALPERN, J, *Zentralb f Chir*, 1922, vii, 519
- ¹⁷ GOSSETT, *Rev de Chir*, 1906, xxxiii, 54
- ¹⁸ GREY TURNER, A, "Pyloroplasty", *Surg Gynecol and Obst*, 1921, June, 537
- ¹⁹ GREY TURNER, A, "Discussion on the Remote Results of the Surgical Treatment of Gastric and Duodenal Ulcers", *Proc Roy Soc Med*, 1920, May, 157
- ²⁰ GRONNERUD, P, "The Etiological Relation of the Sequelæ to Gastro-enterostomy", *Ann of Surg*, 1917, Aug, 177
- ²¹ HOGUET, J P, and COLLE, L G, "Marginal Ulceration after a Modified Polya Operation", *Surg Gynecol and Obst*, 1922, July, 19
- ²² HOHLBAUM, J, *Zentralb f Chir*, 1922, vii, 508
- ²³ HUDDY, G P B, "A Study of the Family Histories of 300 Patients Suffering from Chronic Upper Abdominal Lesions", *Lancet*, 1925, ii, 276
- ²⁴ HURST, A F, and STEWART, M J, "Jejunal and Gastro-jejunal Ulcers", *Ibid*, 1928, ii, 742
- ²⁵ IVY, A C, and FARRELL, J J, "Contributions to the Physiology of Gastric Secretion, etc", *Amer Jour Physiol*, 1925, iv, 639
- ²⁶ JUDD, E S, *Surg Gynecol and Obst*, 1921, xxxiii, 120
- ²⁷ KLEIN, E, *Jour Amer Med Assoc*, 1927, lxxix, 1235
- ²⁸ LEWISOHN, R, "The Frequency of Gastro-jejunal Ulcers", *Surg Gynecol and Obst*, 1925, Jan, 70
- ²⁹ LOURIA, H W, "The Surgical Treatment of Gastric and Duodenal Ulcer", *Ibid* 1928, Oct, 493
- ³⁰ LUFF, A P, "The After-history of Gastro-enterostomy", *Brit Med Jour*, 1929, ii, 1074
- ³¹ MANN, F C, "Experimentally Produced Peptic Ulcers", *Surg Clin N Amer*, 1925, v, 753
- ³² MANN, F C, and WILLIAMSON, C S, "The Experimental Production of Peptic Ulcers", *Ann of Surg*, 1923, April, 409
- ³³ MARTIN, E K, "Post-operative Jejunal Ulcer", *Med Sci Abstract and Rev*, 1923, Jan, 285
- ³⁴ MATTHEWS, W B, and DRAGSTEDT, L R, "The Etiology of Gastric and Duodenal Ulcer", *Surg Gynecol and Obst*, 1933, Sept, 265
- ³⁵ MAYO, C H, "Gastric and Duodenal Ulcers", *Ann of Surg*, 1921, March, 328
- ³⁶ MONTGOMERY, A H, "Gastro-jejunal Ulcer An Experimental Study", *Arch of Surg*, 1923, Jan, 136
- ³⁷ MOYNIHAN, LORD, *Brit Med Jour*, 1919, July 12
- ³⁸ PANNETT, C A, "Debatable Aspects of the Surgery of Gastro-jejunal Ulceration", *Ibid*, 1928, i, 63
- ³⁹ PATERSON, H J, "Jejunal and Gastro-jejunal Ulceration following Gastro-jejunostomy", *Proc Roy Soc Med*, 1909, June, 238
- ⁴⁰ PATERSON, H J, "The Place of Gastrojejunostomy in Gastric and Duodenal Surgery", *Brit Med Jour*, 1926, ii, 555
- ⁴¹ PATERSON, H J, "The Operation of Gastro-jejunostomy and its Physiological Effects", *Lancet*, 1907, ii, 815
- ⁴² RICHARDSON, E P, "Jejunal Ulcer without Previous Gastroenterostomy", *Surg Gynecol and Obst*, 1922, July, i
- ⁴³ SCHOEMAKER, J, "The Results of the Partial Gastrectomy in Cases of Ulcer of the Stomach and Duodenum", *Report of the 8th Congress of the International Society of Surgery*, 1929, i, 20

- ⁴⁴ VON EISELSBERG, F A, "The Choice of Operation in Gastric and Duodenal Ulcer", *Surg Gynecol and Obst*, 1914, Nov, 555
- ⁴⁵ VON EISELSBERG, F A, *Med Press and Circ*, 1914, xcvi, 196
- ⁴⁶ VON HABERER, H, "Termino-laterale Gastroduodenostomie bei der Resektionsmethode nach Billroth II", *Zentralb f Chir*, 1922, xlix, 1221
- ⁴⁷ VON HABERER, H, "Peptic Ulcer of the Jejunum in the Light of Old and Recent Clinical Experience", *Arch f klin Chir*, 1922, cxix, 712
- ⁴⁸ WALTON, A J, "Gastro-jejunal Ulceration", *Lancet*, 1925, ii, 800
- ⁴⁹ WALTON, A J, *A Text-book of the Surgical Dyspepsias*, 1930 London
- ⁵⁰ WALTON, A J, "Primary Jejunal Ulceration", *Brit Jour Surg*, 1922, x, 152
- ⁵¹ WALTON, A J, "Peptic Ulcers Artificially Produced in the Human Being", *Surg Gynecol and Obst*, 1933, June, 997
- ⁵² WALTON, A J, "The Results of Surgical Treatment of Gastric and Duodenal Ulcers", *Brit Med Jour*, 1928, ii, 784
- ⁵³ WRIGHT, GARNETT, *Brit Jour Surg*, 1917, vi, 390
- ⁵⁴ WRIGHT, GARNETT, "Discussion on the Remote Results of the Surgical Treatment of Gastric and Duodenal Ulcers", *Proc Roy Soc Med*, 1920, May, 153
- ⁵⁵ WRIGHT, GARNETT, "Surgical Treatment of Gastro-jejunal and Jejunal Ulceration", *Brit Med Jour*, 1922, ii, 640

PROTRUSIO ACETABULI (CENTRAL LUXATION)

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DURING the last few years considerable interest has been manifested in American literature in the condition of intrapelvic protrusion of the acetabulum, as shown by a number of articles contributed to the journals¹⁻⁶ Prior to this references to the condition are almost entirely restricted to Continental literature, especially the German

Approximately fifty-one true cases have been described in one hundred and ten years since Otto's original article in 1824⁷ The following ten cases are added as likely to be of interest, although there is reason to believe that the condition is not so rare as the above figures would indicate Fifteen cases in all were found in a survey of 298 radiographs of pathological hip-joints, all of which could have been labelled with a broad diagnosis of rheumatic hip—the patients having been sent by their doctors to a rheumatic clinic That is an incidence of 5 per cent in this type of patient

ETIOLOGY AND CLASSIFICATION

In the literature one finds little agreement on the subject of etiology, yet it is mainly on the supposed etiology that attempts at classification have hitherto been made No fewer than sixteen primary causes have been held responsible by the various authors in their reports on cases, and possibly this total is incomplete This has given rise to a wide variation in the ultimate diagnoses

In view of the confusion at present existing, and admitting that protrusion is a disease process, not an entity, I should like to propose a simpler classification, based on the etiology where it is known, but more particularly on the radiographic findings (1) A group conforming to the hypothesis of Eppinger,¹⁴ or some theory of growth disturbance, (2) The rheumatic group, (a) specific infection (gonococcal), (b) non-specific infection, (c) metabolic arthritides, (3) A group including many varieties due to gross destructive disease

Group 1—The infantile acetabulum is the site of union of the three pelvic bones, by the medium of a tri-radiate cartilage, in which ossific centres appear in the twelfth year and union commonly takes place about the sixteenth year

Eppinger considered that the cases he described were due to a disturbance of growth affecting this Y-shaped cartilage, possibly a process resembling the more recently described osteochondritis

The etiology of this process of malacia has not been explained satisfactorily There is no direct evidence to support this theory, but equally it is not likely to be disproved

On the evidence of the history and radiographic findings, I suggest that, for a small group, Eppinger's theory, which has never been accepted, is correct

The radiograph of this type shows a joint with an intact cartilage and no evidence of infective changes in articular bone. The acetabulum protrudes to a varying extent, and on this depends the degree of limitation of movement, which may be so slight that the patient is not aware of defect,¹⁵ or, alternatively, the loss of full range of movement, especially abduction, which she notices, is insufficient to make her seek treatment (see *Figs 24, 25*). At a later stage, owing no doubt to the mechanical disadvantage at which the joint is working, osteo-arthritic and infective changes are often superimposed, and the patient now complains of definite pain, or, by reason of these changes, which produce osteophytes and loss of cartilage, or both, the patient is aware, for the first time, that movement is restricted.

The histories which *Cases 1* and *2* gave also point to a non-inflammatory origin in early life —

Case 1 —The patient gives a clear history of limitation of movement since the age of 15, she remembers that when she was learning to swim the instructor remarked on the fact that she was unable to separate her legs so as to perform the 'frog-leg' kick of the breast stroke—her legs "trailed behind her", she had no pain at this time. For the last twenty years, however, she has had pain in the knees and the lower back, which may have been referred from the hips. She first noticed definite pain in the left hip four months ago after a fall. The condition is bilateral, but she has no pain on the right side (*Fig 24*).



FIG 24 —*Case 1*. Left hip. Female, age 62 years. Bilateral. Duration forty-seven years. Limited movement forty-seven years. Pain in left hip four months. Right hip no symptoms except limited movement. No pain on this side, but condition identical. Cartilage intact. No arthritic changes. No other joints involved (*Case of Dr Copeman*).



FIG 25 —*Case 2*. Left hip. Female, age 40 years. Bilateral. Duration twenty-four years. Limited movement twenty-four years. Pain in right hip, and to a lesser extent in left hip, for nine years. Cartilage thin at the point of pressure, otherwise intact. Floor of acetabulum extremely thin. No evidence of infective changes in bone or joint. Osteophyte formation commencing. No other joints involved (*Case of Mr Verrall*).

Case 2 —The patient noticed she was unable to obtain the full movements of the hip-joints when 16, she then found she was unable to cross her legs when seated, and she has been aware of limitation of movement since that time (*Fig 25*).

Case 3 —This case is placed in this group on radiographic evidence alone. The patient first noticed pain and stiffness in the hips four months ago, she was not aware of limited movement before this. This might be explained by the fact that, although the head is protruding, the neck has not been burned. Radiographically, the most obvious obstruction to movement is the presence of osteophytes on the femoral head, and it is difficult to imagine that these have developed in four months. Otherwise, this case resembles *Case 2* (*Fig 26*).

Amongst others, the cases of Frolich¹⁵ could be classified in this group. Similarly Hertzler¹⁶ has reported a case aged 29 years with a history of ten years' duration. Recently Reed⁶ has published the case of a nurse aged 24 years with the onset of disability at 16 years.

The conclusion is, therefore, that there is a small group of cases which is compatible with a mode of origin such as Eppinger has described, or with some similar theory.

The protrusion, having formed, does not appear to increase in size. *Case 2* is in substantially the same condition as in 1929,³ except that osteophytes are now forming and the cartilage on the right side is becoming thin, the protrusion, however, is no greater. In view of the presence of an intact cartilage in the early stages and the absence of sclerosis of bone or other signs of infective changes, the

FIG 26—*Case 3*. Left hip. Female, age 33 years. Bilateral. Duration stated as four months. Onset of pain and limitation of movement simultaneously four months ago. Acetabulum the thickness of paper. A very large osteophyte has formed on the head of the femur. No other joints involved. Similar condition to *Case 2*. (*Case of Dr Heald*.)

term 'arthrokatadysis' of Verrall is very suitable for the group. Later the combination of trauma and superimposed infection, to which the joint in this state appears susceptible, destroys the characteristic features, and it is not possible to differentiate this group from the following.

SUMMARY—Summarized, these points are—

1 A history of limited movement, especially abduction and adduction, the onset being in adolescence and painless.

2 Since the limitation of movement depends on contact being made on the rim of the acetabulum by the femur, the minor varieties may be overlooked, until rim osteophytes further limit movement or infection causes pain.

3 The protrusion appears to be non-progressive, the cartilage is of normal width, and the acetabulum floor very thin. The articular bone is unaltered in appearance. Later, infective arthritic changes tend to be added, these changes are progressive.

Group 2 (Rheumatic Group)—

Certain evidence has been produced in the past which favoured the gonococcal origin of some of these cases, notably that found in the writings of



FIG 27—*Case 4*. Left hip. Male, age 42 years. Unilateral. Duration eighteen years. Circumferential thinning of cartilage. Sclerosis of femur head and acetabulum. Secondary osteophytes. No other joints involved. In spite of the radiographic appearance, the patient denies any hip trouble before the acute onset—was a regular member of a football team and had played all games.

Schlagenhauser,⁸ Henschen,⁹ Kienbock,¹⁰ Breus,¹¹ Chiari,¹² and Zwicker¹³ Of the five male patients in this series, four had had gonorrhœa From this point of view the histories are summarized —

Case 4 —Age 42 years First noticed pain in the left hip at 24 years of age, acute onset of severe pain, was in hospital for five months, had contracted gonorrhœa eight weeks before onset of pain, no previous disability of hip No other joints involved Unilateral (*Fig 27*)

Case 5 —Age 63 years Pain began in 1916 in the left hip It was of moderate severity, and the first attack lasted a week, it then became intermittent He was admitted to hospital in 1919 for seven weeks for hip trouble Noticed pain in the other (right) hip four years ago Had gonorrhœa twice between 1904 and 1909 Bilateral changes No other joints involved (*Fig 28*)



FIG 28 —*Case 5* Left hip Male, age 63 years Bilateral Duration seventeen years left hip, and four years (?) right hip Dense sclerosis of femur head with cavitation of bone, the outline is irregular and pointed Osteophytes present More recent radiographs show loss of cartilage No other joints involved (*Case of Dr Nisse*)



FIG 29 —*Case 6* Right hip Male, age 64 years Bilateral Duration forty-four years Sclerosis of femur and cavitation Thickened and sclerosed acetabulum Cartilage absent except outer and upper aspect of joint (*Case of Dr Ray*)

Case 6 —Aged 64 years Pain in hips at 20 years of age, limped slightly when walking, no acute attacks, constant pain now Gonorrhœa at 20 years Bilateral No other joints involved (*Fig 29*)

Case 7 —Aged 67 years Pain began about seven to eight years ago, as far as he can remember, was first noticed after a minor fall Had gonorrhœa in 1925, and is being treated at present for stricture Is not sure of the time relation between infection and hip pain, is mentally dull and unreliable Bilateral No other joints involved (*Fig 30*)

The coincidence of arthritis with venereal infection in these patients suggests that the latter may have had some casual relationship to the joint lesions

Case 8 (the fifth male in the series) —The patient was a man aged 44 years, with very advanced spondylitis of the so-called Marie-Strumpell type, with complete calcification of the intervertebral ligaments and obliteration of the sacro-iliac synchondrosis There was no history of gonorrhœa, and the condition was bilateral (*Fig 31*)

In the histories of the females of the series the gonococcus could not be excluded at this late stage with any certainty, few definite factors were discovered which appeared to bear direct relationship to the hip disease One case suffered

from polyarthrititis, the onset being at the menopause. Radiographs of many joints were obtained and the appearances were those of a metabolic rather than an infective arthritis. Two cases (*Figs 32, 33*), aged 67 and 76, had a subacute onset with multiple joint involvement and were considered non-specific infections.

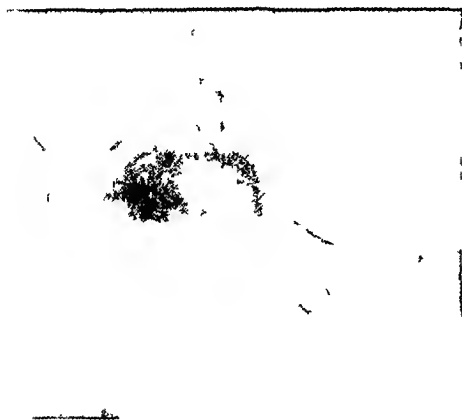


FIG 30—*Case 7* Right hip Male, age 67 years. Bilateral. Duration about eight years. Femur head dense with irregular outline and has protruded to the limit of the neck. The cartilage is very thin. The floor of the acetabulum is sclerosed. Osteophytes are present. (*Case of Dr Copeman*)



FIG 31—*Case 8* Left hip Male, age 44 years. Bilateral. Duration ten years. Onset of spondylitis twenty years ago. Cartilage thinned. Little or no reaction in bone of head of femur. Osteophytes present. Calcification of intervertebral ligaments and obliteration of sacro-iliac synchondrosis. (*Case of Dr Ray*)



FIG 32—*Case 9* Left hip Female, age 67 years. Unilateral. Duration stated as one year. Head of femur sclerosed and osteophytes present. Cartilage absent except outer aspect of joint. Acetabulum sclerosed. In left knee and right wrist pain and swelling. Gross dental sepsis. (*Case of Dr Howitt*)

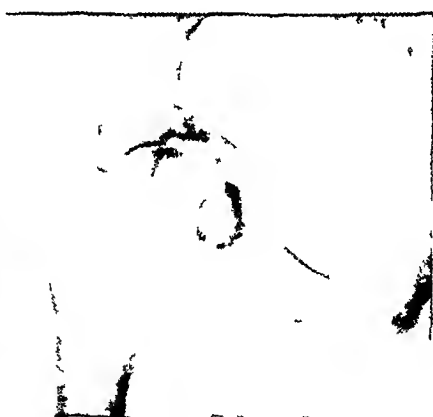


FIG 33—*Case 10* Right hip Female, age 76 years. Unilateral. Duration one year. Irregularity of articular surface of femur. Sclerosis and thickening of acetabulum. Generalized loss of cartilage. Osteophytes developing. Pain and swelling of left wrist two to three years. Gross dental sepsis. (*Case of Dr Nisse*)

From the evidence of these cases, it can be inferred that the etiology of this group is the etiology of chronic rheumatic affections in general, and a specific cause to explain all cases is lacking.

It is not possible to describe an exact picture that will include all the characteristics of this rheumatic group, it is typical, however, that the cartilage is absent or thin, and this thinning is generalized over the circumference of the femur head, not at the point of maximum pressure only, as in early senile osteo-arthritis. The head of the bone is often heavily sclerosed, with minor irregularities in outline, and occasionally somewhat pointed. New bone is laid down as a compensatory process over the bulging acetabulum, a feature which is absent from *Group 1*, except in the later stages. The wall is therefore more thickened and dense in *Group 2* than in *Group 1*.

Possibly the degree of protrusion in *Group 2* depends to some extent on the virulence of the infective organism. Similarly, it would seem to be necessary for these infective changes in the acetabulum to be in advance of any destruction which may take place in the femur head. *Case 4* shows that a virulent infection alone will not necessarily produce marked protrusion, in this patient a softened and flattened femur head has made the result less marked than otherwise might have been expected. Osteophytes develop in this form of the disease, especially at the base of the femur head. The head may itself show cavitation, as is found so frequently in the common osteo-arthritic changes (*morbis coxæ senilis*) which affect this joint.

There is now a tendency to classify all cases of intrapelvic protrusion as osteo-arthritis. This is correct only as a statement of end-result. Admittedly in some cases osteo-arthritis is the only possible diagnosis, occasionally, however, by a careful history and correlation with X-ray appearances, the origin of the deformity may be traced.

In some cases of this series the fact has been observed that little reliance can be placed on the patient's estimate of the duration of the arthritis when the history given is short. Many of the changes in the radiographs could not possibly have been produced in the brief period of the patient's complaint. *Case 1* considered her trouble began four months ago, when she first suffered pain. She was neglecting the fact that she had had limited movement for nearly fifty years.

Group 3—The third group is a heterogeneous collection of little interest, the condition being often something in the nature of an accidental occurrence in some gross disease of bone, as for example when secondary neoplasm²⁰⁻²³ or echinococcal²⁴ disease of bone involves the acetabulum, or a part of the destruction produced by pyogenic organisms,²⁵ syphilis,²⁶ and tuberculosis.^{27, 28}

It would simplify descriptions if this group were excluded from the reports of true Otto pelvis.

I am greatly indebted to the Physicians of the British Red Cross Clinic for Rheumatism, Drs Copeman, Heald, Howitt, Nisse, and Ray, for the generous manner in which they have allowed me to use their cases, to Mr Verrall for permission to radiograph his case, and to Dr Gilbert Scott for the use of the radiographic material of the Clinic.

REFERENCES

- ¹ POMERANZ, M. M., "Osteo-arthritic Protrusion of the Acetabulum", *Arch of Surg*, 1922, v, 691.
² DOUB, H. P., "Intrapelvic Protrusion of the Acetabulum", *Radiology*, 1929, xii, 369.

- ³ VERRALL, P J, "An Unusual Bilateral Condition of the Acetabula", *Jour Bone and Joint Surg*, 1929, xii, 30
- ⁴ CARY, N A, and BERNARD, L, "Arthrokata-dysis of the Hip-joint", *Ibid*, 1932, xiv, 687
- ⁵ POMERANZ, M M, "Intrapelvic Protrusion of the Acetabulum (Otto Pelvis)", *Ibid*, 1933, 663
- ⁶ REED, EDWARD N, "A Case of Arthrokata-dysis of the Hip-joint", *Ibid*, 1933, July, 802
- ⁷ OTTO, A W, "Pfannenbeckenmissgestaltung Infolge deformierender Osteo-arthritis in neue Seltene", *Beobachtungen sur Anatomie, Physiologie und Pathologie gehorig*, 2nd ed, 1824, 19 Berlin A Rucker
- ⁸ SCHLAGENHAUFER, F, "Ueber Coxitis gonorrhoeica und ihre Beziehungen zur Protrusion des Pfannenbodens", *Zentralb f Gynakol*, 1909, xxxiii, 228
- ⁹ HENSCHEN, K, "Die pathologische (spontane) Luxatio centralis Femoris", *Beitr z klin Chir*, 1909, lxxv, 599
- ¹⁰ KIENBOCK, R, "Ueber die mit Protrusion des Pfannenbodens Einhergehenden Erkrankungen des Huftgelenks und ihre Beziehungen zur Arthritis gonorrhoeica und Arthropathie bei Tabes", *Fortschr a d Geb d Rontgenstrahlen*, 1912, xviii, 280
- ¹¹ BREUS, C, "Zur Aetiologie und Genese der Ottoschen Protrusion des Pfannengrundes", *Wien klin Woch*, 1913, xxxvi, 167
- ¹² CHIARI, H, "Ueber die Aetiologie und Pathogenese der intrapelvinen Pfannenprotrusion", *Beitr z klin Chir*, 1916, cii, 318
- ¹³ ZWICKER, "Ueber Pfannenbodenvorwölbungen im Rontgenbild", *Forts a d Geb d Rontgenstrahlen*, 1927, xxxvi, 1008
- ¹⁴ EPPINGER, H, "Pelvis Chrobak Kovarthrolithesisbecken" (Festschr f Chrobak) *Beitr z Gebh u Gynakol*, 1903, ii, 176
- ¹⁵ FROLICH, M, "Migration intrapelvienne progressive de la Tete femorale et Lesion de l'Ovaire", *Rev d'Orthop*, 1930, xvii, 553
- ¹⁶ HERTZLER, A E, "Osteo-arthritic Protrusion of the Acetabulum (Intrapelvic Pfannenprotrusion)", *Arch of Surg*, 1922, v, 691
- ¹⁷ THOMSEN, F, "Krebsige Osteomalacie", *Arch f klin Chir*, 1872, viii, 237
- ¹⁸ HERTZLER Ref 16 Case 4
- ¹⁹ SENFTLEBEN, *Arch f klin Chir*, 1860, i, 81
- ²⁰ VALENTIN, B, and MULLER, H, "Intrapelvine Pfannenprotrusion", *Ibid*, 1921, cxvii, 523 (Case 1)
- ²¹ TRENDLENBURG, R, "Echinococcus multilocularis der rechten Beckenhälfte", *Verhandl d deut Gesellsch f Chir*, 1881, v, 60
- ²² ESAU, P, "Akute Osteomyelitis des rechten Schenbels und zentrale pathologische Luxation des Oberschenkels", *Deut Zeits f Chir*, 1907-8, xci, 611
- ²³ FERE, C, "Description de quelques Pieces relatives aux Lesions osseuses et articulaires des Ataxiques conservees au Musee anatomo-pathologique de la Salpetriere", *Arch de Neurol*, 1882, iv, 202
- ²⁴ KIENBOCK, R Ref 10 Case 1
- ²⁵ CHIARI, H Ref 12 Case 8
- ²⁶ VALENTIN, B, and MULLER, H Ref 20 Case 2
- ²⁷ HERTZLER, A E Ref 16 Case 3
- ²⁸ CHIARI, H Ref 12 Cases 3 and 4

BONE-GRAFT FOR NON-UNION OF THE CARPAL SCAPHOID*

By GORDON MURRAY, TORONTO

WHEN a patient complains of a 'sprained wrist' with symptoms lasting more than two weeks, the possibility of a fracture of the carpal scaphoid should be considered and the necessary investigation undertaken. If examination shows a fair range of movement of the wrist-joint in all directions, but limitation at the extremes of all movements, with tenderness in the anatomical snuff-box when the hand is adducted, and tenderness over the dorsal and palmar surfaces of the scaphoid, with negative findings elsewhere, it is probable that there is a fracture of the scaphoid. If after an injury causing symptoms in the wrist-joint these findings persist for months or years, the possibility of non-union of a fractured scaphoid should be considered.

X-rays taken of the carpal scaphoid in the antero-posterior and oblique directions usually provide the necessary evidence, but recent fractures may be difficult to see with X-rays taken in various directions. There are cases of recent fracture in which a clinical diagnosis is easily made but X-rays do not show the lesion at that time, yet plates taken a few weeks later show obvious signs of fracture. Older fractures with non-union are seen more easily, owing to the early rarefying osteitis and fibrous union, and later sclerosis of the adjacent margins of the fragments.

Fractures of the tuberosity always unite if the fragments are in apposition, and those through the intra-articular surfaces about the waist of the bone, which are the most common, may unite if treated. Johnson¹ has shown experimentally that there is an adequate blood-supply to the bone as a whole, and to both fragments, in case of fracture. He showed also that the type of reaction in fracture of the scaphoid is similar to that in other bones, but is more localized to the fracture line, as there is so little periosteum to assist in the formation of subperiosteal callus. The formation of bone in the callus is slower than in other fractures.

Adams and Leonard² say that untreated fractures of the scaphoid always result in non-union, and with the usual form of treatment a small percentage shows union. Grace³ supports this view with a report of cases showing a high percentage of non-union in those treated by immobilization for a period varying from six to eight weeks. Bohler,⁴ on the other hand, states that most fractures of this bone unite if treated early by prolonged fixation, and this agrees with our results. However, there are many injured wrists treated as sprains without fixation which provide the cases of fracture with non-union.

Certain anomalies should be kept in mind to prevent confusion in making a diagnosis of fracture and non-union. Ordinarily the centre of ossification appears in the sixth year, but there may be two centres and these may not unite, but may persist to form two bones. Rarely the os centrale may persist as a third bone.

* From the Department of Surgery, University of Toronto

Methods of treatment that have been advocated for non-union are excision of one or of both fragments or of all the bones in the proximal row, including the fragments of the scaphoid. Any one of those operations leaves a deformed wrist with some permanent disability in the form of impaired function and frequently with pain on active use of the hand (Grace³).

If the individual with non-union of a fractured scaphoid is able to follow a sedentary occupation and avoid energetic use of the hand, he may have little discomfort, but if heavy labour, sport, or full range of movement of the wrist is important, the best prospects are offered by a bone-graft. There is a report of one case² in which this operation was used, but it has not come into general use. In our cases with non-union this form of treatment has yielded the best results. It is essential to have X-ray evidence that both fragments are viable and in apposition, and that there is no arthritis.

OPERATIVE TECHNIQUE

With the hand in full adduction, a curved incision is made along the radial surface of the wrist-joint extending about $1\frac{1}{4}$ in upward and downward from the radial facet of the scaphoid. The ends of the incision are curved towards the posterior surface of the wrist, and the convexity anteriorly should reach the tendon of the abductor pollicis longus. The radial nerve and vessels and the abductor tendons of the thumb are retracted anteriorly, and the extensor pollicis longus tendon posteriorly. This provides exposure of the tuberosity of the scaphoid. A small transverse opening is made through the dorsal capsule of the wrist-joint, exposing the dorsal surface of the radial facet of the scaphoid, and on this surface the fracture line is apparent. If the other bones of the carpus have not been disturbed by the injury, the fragments of the scaphoid will not be displaced, and in that case the fracture line is not disturbed by curetting, etc. After clearing the most prominent area of the tuberosity, a small nick is made in the bone at this point with rongeurs, in order to provide for countersinking of the graft and prevention of bone proliferation, which might interfere with abduction of the wrist-joint.

With a bit about $\frac{1}{16}$ in a hole is drilled, beginning at the nick in the tuberosity, through the proximal fragment, across the fracture line, and into the distal fragment. Great care is necessary to line the drill properly, assisted by observations through the dorsal window, so that no cartilaginous surface is damaged. The depth of the drill hole, after the fracture line is crossed, should be measured every few millimetres to prevent damage to the semilunar facet of the scaphoid by going too far.

A suitable piece of cortical bone is removed from the tibia and shaped to fit snugly. It is passed well through into the medial fragment, taking care that the fragments are not separated, and is cut to leave no projection. The dorsal ligament is repaired.

The hand is supported in a circular plaster in a cock-up position for eight weeks. After this period all our cases in the general hospital had X-ray evidence of bony union, and within a few months the fracture line had disappeared. There was complete restoration of function, with a full range of movement in all directions without pain and with normal grip.

NON-UNION OF CARPAL SCAPHOID

65

CASE REPORTS

Case 1—B S, aged 31, painter Admitted on Jan 27, 1931, with a complaint of pain and stiffness of the right wrist for two months This began with an injury received when cranking a car, and persisted with no improvement of symptoms A diagnosis of sprained wrist was made by his family physician, and was treated by bandaging

On examination there was no deformity and no swelling There was limitation of flexion, extension, adduction, and abduction, with marked pain on active and passive movements beyond this range of movement The grip was weak, and increased force applied in any way caused pain in the wrist region With the hand adducted there was marked tenderness over the radial facet of the scaphoid, just distal to the styloid process of the radius X-rays (*Fig 34*) showed a fracture through the waist of the scaphoid, without displacement, and both fragments apparently viable

A bone-grafting operation was undertaken, and *Fig 35* shows the graft in place, bridging the fracture line and lying in contact with both fragments *Fig 36*, taken four months after operation, shows solid union of the graft to both fragments and disappearance of the fracture line The circular plaster extending from the elbow region to the metacarpo-phalangeal joints was replaced in ten days by a more snugly fitting one, which was left on for eight weeks X-rays taken at this time showed signs of union but firm union as shown in *Fig 36* was evident after a period of four months The post-operative course was uneventful Limited activities of the wrist-joint were allowed



Fig 34—*Case 1* X-ray taken two months after fracture of carpal scaphoid, showing non-union

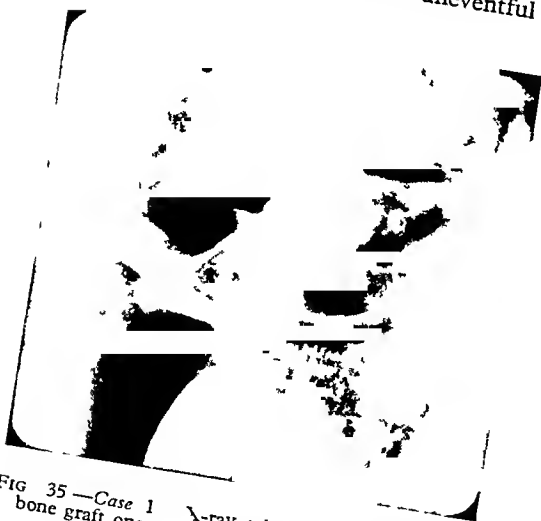


Fig 35—*Case 1* X-ray taken immediately after bone graft operation, showing graft in position



Fig 36—*Case 1* X-ray taken four months after bone-graft, showing solid union of fragments

after removal of the cast at eight weeks, and full range of movement was present without discomfort after fourteen weeks The patient returned to his usual occupation of painting and decorating four months after operation On clinical examination there was no disability of the wrist-joint, and this has remained unchanged over a period of two and a half years

Case 2—F S, aged 23, medical student. Admitted on June 2, 1931, with pain and disability of the right wrist-joint of four months' duration. A fall during a game of rugby caused some pain and stiffness of the wrist-joint which persisted. An X-ray taken on the day of the injury did not give conclusive evidence of a fracture. After four months' treatment by bandages and wrist supports by the family physician, X-rays showed a fracture with well-marked rarefying osteitis, with both fragments viable, as shown in *Fig 37*. On June 4 a bone-graft was placed across the fracture line. The small proximal fragment made the operation technically more difficult, but it was carried out without injury to articular cartilage (*Fig 38*). The wrist-joint was fixed in plaster for eight weeks, and in four months solid union of both fragments and graft had taken place (*Fig 39*). There was complete recovery of function of wrist-joint, with full range of movements in all directions without discomfort, and the patient was able to return to regular sports on Dec 1, 1931.

Case 3—H C, aged 32, fireman. Feb 20, 1932, a fall on the outstretched left hand caused severe injury to the wrist. This was treated by his family physician as a sprained wrist for about one month without improvement in symptoms. Examination at this time showed no deformity, no swelling, but very great limitation of all movements, and tenderness in the anterior, posterior, and lateral surfaces of the scaphoid. X-rays showed a typical fracture through the waist of the bone, with no displacement, with both fragments viable, and with no signs of union.

At operation on March 24 a small groove was cut across the fracture line, which showed no signs of union, and a small graft of bone from the tibia was fitted in place (*Fig 40*). After fixation for eight weeks, the plaster was removed and X-rays were taken. There was some X-ray evidence of union, so the arm was left without support and movements were encouraged. The graft and fragments united solidly and a full range of movement without discomfort was obtained. The patient returned to his regular occupation as fireman without disability, and has continued at this work for more than one year.

Case 4—F P, aged 33, labourer. On May 18, 1932, both wrists were injured, a Colles's fracture of the left, and a fracture of the scaphoid on the right. With the usual treatment the Colles's fracture united and a good result was obtained, but the scaphoid continued to cause trouble. Light work was resumed for a short period during September and October, but was discontinued on account of pain in the right wrist. Examination on Dec 21 showed some swelling in the region of the wrist-joint with painful limitation of all movements, and tenderness over all surfaces of the scaphoid. X-rays showed a fracture through the waist of the bone, without displacement, with no signs of union, and with both fragments viable.

At operation by Dr W E Gallie on Dec 22 an incision across the anatomical snuff-box exposed the scaphoid with the fracture line. There was firm fibrous union, but no evidence of bony union of the fragments. A drill-hole was made to cross the fracture line and a graft from the tibia fitted in place. The fracture surfaces were not disturbed.

Primary healing occurred, and the plaster support was removed on Feb 23, 1933. X-rays showed evidence of union of the graft and both fragments (*Fig 41*). Further X-ray plates on May 14 showed more union by bone. The patient returned to his work as a labourer on May 15, and was able to carry on without discomfort.

Case 5—J M, aged 37, labourer. Admitted on Oct 1, 1932, with a history of a fall on June 8, resulting in a fracture of the distal end of the radius, with anterior dislocation of the semilunar bone and fracture of the carpal scaphoid. The other injuries had been treated successfully, but there was non-union of the fracture of the scaphoid. There was limitation of all movements of the wrist, including rotation, with weakness of the grip, and partial anæsthesia of the ulnar nerve distribution in the hand. Some swelling was present over the lower end of the radius and carpal bones, with fairly marked tenderness over the dorsal and palmar surfaces of the scaphoid. X-rays at this time showed union of the fracture of the radius, normal position of the semilunar, and a non-union of a fracture through the waist of the scaphoid, with both fragments viable.

Operation was performed on Nov 1 by Dr W E Gallie. The fracture surfaces were cleared and freshened, and on account of the direction and position of the fracture, it was thought to be easier to cut a groove in the distal fragment, through the ligamentous area,



FIG 37—Case 2 X-ray taken four months after injury, showing non-union of scaphoid



FIG 38—Case 2 X-ray taken after operation, showing the bone-graft in position



FIG 39—Case 2 X-ray taken four months after bone-graft, showing solid union of fragments



FIG 40—Case 3 X-ray taken following operation, showing bone-graft in position



FIG 41—Case 4 X-ray taken eight weeks after operation showing union of bone-graft of both fragments, and fracture line disappearing



FIG 42—Case 5 X-ray taken twelve weeks after bone-graft, showing union of graft and fragments

and to drill a hole in the proximal fragment. A graft from the tibia fitted nicely into both fragments, and the fracture surfaces remained in fairly good apposition.

A circular plaster was applied. The post-operative course was not complicated and the plaster was removed on Jan. 4, 1933. X-rays taken at that time showed the graft in good position, with signs of union of the graft to the distal fragment. There was free and painless movement of the wrist-joint. An anterior cock-up splint was applied for four weeks, at which time X-rays showed firm union of the fragments to each other and to the graft (*Fig. 42*). Movements of the wrist were very good, and the patient was able to return to light work on March 15, and to heavy work on May 1.

Since submitting this paper for publication, four other cases have been treated successfully by this method.

CONCLUSIONS

- 1 Recent fractures of the carpal scaphoid may be difficult to see on X-ray films, but later are seen easily.
- 2 Many cases if treated early will unite by bone, but if neglected may develop non-union.
- 3 Non-union treated by bone-graft has given excellent results with complete recovery of function in our cases.

REFERENCES

- ¹ JOHNSON, Jr, R. W., *Jour. Bone and Joint Surg.*, 1927, July.
- ² ADAMS, J. D., and LEONARD, R. D., *New Eng. Jour. Med.*, 1928, April 12.
- ³ GRACE, R. V., *Ann. of Surg.*, 1929, May.
- ⁴ BOHLER, L., *Treatment of Fractures*, 1929.

X-RAY INVESTIGATION OF THE UPPER RIGHT QUADRANT*

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WITH the exception of the introduction of the barium meal as an aid in diagnosis of lesions of the gastro-intestinal tract, no single contribution for the application of opaque media in radiological diagnosis has been as valuable as the work of Evarts A Graham and his collaborators in developing and perfecting the use of sodium tetraiodophenolphthalein to demonstrate hepatic function and cholecystic disease

One evening in the winter of 1922, Dr Graham, who had given up his surgical work in 1913 and 1914 for the study of chemistry, was ruminating on the work of Abel and Rowntree which had demonstrated the almost complete excretion of chlorinated phenolphthalein through the bile. He conceived the idea that, by attaching atoms of some substance opaque to X rays to the phenolphthalein in place of the chlorine, it might be possible to obtain a shadow of the gall-bladder. Casting around for some such substance, he noted in a list of indicators supplied by the Eastman Company the free acid of tetraiodophenolphthalein. This indicator was obtained and Dr Warren Cole converted it into the sodium salt on account of its greater solubility before injecting it intravenously into their experimental dogs. Six dogs were injected, and on the following morning when X-ray films were made five of the dogs showed no shadow in the gall-bladder, and one of them showed the gall-bladder faintly. On investigation it was found the animal keeper had forgotten to feed the dog in which the shadow of the gall-bladder was obtained and had fed the other five. If that keeper had been utterly efficient, we might not have had the art of cholecystography as it exists to-day!

From that time onward numerous other salts—forty-eight in all—were tried. Of these, thirteen gave gall-bladder shadows, but they all possessed disadvantages, and eventually, with better chemical preparation, the original salt of sodium tetraiodophenolphthalein was again utilized, and it is the basis of all the dyes used to-day, either orally or by intravenous injection.

Controversy for some time was rampant over the proper method of administration, whether the dye should be given by mouth or intravenously. Even to-day each side has its advocates, but the oral method has far out-distanced the intravenous one on account of its easier administration, slighter systemic reaction, and absence of danger of the serious results that may occur at the site of injection if given intravenously, and also in a well planned carefully carried out oral technique the percentage of correct diagnoses of cholelithiasis and cholecystitis is equal to that obtained by the intravenous method. During the last seven years we have used the oral method exclusively and find no reason to employ the intravenous

* Read before the Section of Medicine, Academy of Medicine, Toronto, on Oct 10, 1933

INDICATIONS AND CONTRA-INDICATIONS

The possibility of the presence of gall-bladder disease should be investigated in all patients complaining of pain or distress in the upper abdomen. For this investigation cholecystography should be used, but may we urge, and urge most strongly, that the gall-bladder investigation should not be the only examination? An opaque meal should always follow after cholecystography, and some cases will call for the administration of a barium enema, or the intravenous use of the colloidal solution of thorium dioxide, before a clean-cut diagnosis is obtained of the actual lesion present which simulates cholecystitis or cholelithiasis. The examinations should be carried out in the order named above. If the meal, enema, or thorium is administered first, the cholecystogram will be obscured by barium in the bowel or thorium in the liver.

Many conditions have been mentioned by other investigators as contra-indications to the administration of the dye. In our laboratory our experience has been that the only contra-indications are chronic increasing jaundice, acute jaundice, myocardial degeneration, and advanced debility. In the chronic increasing jaundice we have never been able to fill the gall-bladder, and our line of investigation has been to rule out the presence of carcinoma of the pancreas, of the ampulla of Vater, or of the gall-bladder, by the barium meal or thorium dioxide, or the presence of a stone or obstruction of the common duct by flat films of the gall-bladder region. If the dye is administered in acute jaundice, serious damage to the liver cells may result, and several fatalities have been reported from such a procedure. When myocardial degeneration is present the systemic reaction from the exhibition of the dye may cause such a serious upset that a fatality may result. The same applies to the exhibition of the dye in advanced debility.

In secondary carcinoma of the liver where the primary is in the gastrointestinal tract, in syphilis of the liver in its various forms, in hypertrophic cirrhosis, and in polycystic disease of the liver we have administered the dye with no ill effects, and have frequently been amazed at the presence of apparently normal function with a large amount of hepatic involvement present.

METHOD OF ADMINISTRATION OF THE DYE

The oral administration of the dye calls for intelligent co-operation by the patient if accurate results are to be obtained. Often we found on inquiry that the lack of filling of the gall-bladder was due, not to disease, but to non-attention to some of the instructions given to the patient.

It is wise to have the colon empty twenty-four hours before the examination is carried out, castor oil or compound liquorice powder being the most suitable laxatives. The salines leave a large amount of gas in the colon which may overlie the gall-bladder region and simulate cholesterol stones with a calcium periphery.

On the evening of the administration of the dye the patient takes a large meal at 7 o'clock consisting of the following: bread, but no butter; vegetables, but no butter, oils, or fat; salads without oil; fresh fruit and jams—and only water to drink. At 8 o'clock the dye is taken and the patient immediately retires. The following morning we make the first examination at 9 o'clock with the patient fasting and only allowing enough time to dress and come to the laboratory. Even

the odour of food should be avoided, as we have been able to empty a normally filled gall-bladder by passing a freshly prepared dish of bacon and eggs under the patient's nose. Occasionally a gall-bladder is slow to fill, and if this occurs a second observation should be made in two hours, i.e., fifteen hours after the ingestion of the dye. Then a fat meal is given and the last examination made at eighteen hours after the ingestion of the dye. If the gall-bladder filled well at the first examination, the second one may be omitted, the fat meal taken at once, and the examination for emptying made at the eighteenth hour. The question is often asked: If vomiting occurs, does it render the examination useless? In our experience we have found that vomiting taking place thirty minutes after drinking the dye has no effect upon a satisfactory observation, because within that time sufficient of the sodium tetraiodophenolphthalein has passed to be absorbed within the intestinal tract and give a satisfactory concentration within the gall-bladder if it is normal. The same answer applies to the attacks of diarrhoea occasionally occurring with the exhibition of this salt. Unless the diarrhoea is extremely severe and manifests itself within a short time, it will have no effect upon the end-result. If dye is observed in the cæcum and ascending colon at the first observation, we conclude the patient has absorbed sufficient to show a well filled gall-bladder if this viscus is functioning normally.

The dye is absorbed from the digestive tract, excreted by the liver, passes to the gall-bladder, where it is concentrated by the mucosa along with the normal bile, and from there it passes through the cystic and common ducts again into the digestive tract.

It is not necessary to go into full detail of all the radiological technique—suffice it to say a kilovoltage as low as will completely penetrate the individual should be used, thus obtaining a soft film with a wealth of detail. The milliamperage will be governed entirely by the length of time needed for the exposure. Numerous films should be made, and the whole area from the 9th rib to the lower portion of the sacro-iliac joint should be covered if a thorough demonstration of the gall-bladder area is to be made.

POSITION OF THE GALL-BLADDER

The gall-bladder follows the same rules of habitus as do the viscera so well demonstrated by the late Dr. Walter Mills, of St. Louis. He divided the types of habitus into four, and gave us the hypersthenic, sthenic, hyposthenic, and asthenic classifications. In the hypersthenic individual the gall-bladder is found high, level with the 10th to 12th ribs, well out from the spine and lying in an almost transverse position. In the sthenic and hyposthenic type it is placed more obliquely, situated at a lower level and closer to the spine, while in the asthenic individual it will be level with or below the iliac crest and close to or superimposed upon the spinal column.

NORMAL FILLING

The normal gall-bladder commences to fill in eight hours, is well filled in thirteen, and the greatest concentration occurs about the sixteenth hour after ingestion. It should be empty three hours after the ingestion of a fat meal. In the sthenic to the asthenic type it is pear-shaped, and we divide it into the fundus,

body, and neck. In the hypersthenic it lacks this characteristic and is practically oval in appearance. The average content in all types is about one and a half fluid ounces. The mobility varies in the different types, is least in the hypersthenic and most mobile in the asthenic type. One must never forget the possibility of a situs transversus when the shadow is not seen on the right side, and inspection of the left side of the film should be made to rule out this possibility. Generally speaking, a gall-bladder that fills well at the first observation, shows contraction and increased concentration at the second examination, and is empty three hours after the fat meal, may be considered normal (*Figs 43, 44*).



FIG 43—Normal gall bladder showing concentration of the dye at the first examination



FIG 44—Normal function of gall bladder containing cholesterol stones. These stones would probably be missed if the concentration of the dye had not occurred

ABNORMAL FILLING

While the gall-bladder may be filled well and empty well, the smooth pear- or oval-shape may show some deformity, and it is well to bear in mind that this may be due to extrinsic pressure before any statement of pericholecystic adhesions is made. Before the latter can be established, the deformity in contour should be present on the films of a subsequent examination, and, as in duodenal ulcer or carcinoma of the stomach, should show no change during the first or subsequent observations. In some cases a constant deformity in a well filled gall-bladder is due to a small ulcer of the mucosa accompanied by muscular contraction similar to the spasmodic incisura noted in gastric ulcer, or it may be present after such an ulcer has healed like the organized hour-glass deformity in the stomach (*Fig 45*).

During the examination the gall-bladder may be fairly well filled, and at the subsequent examinations the viscus is slow to empty. In our opinion this points towards a chronic inflammatory change, the mucosa being damaged (*Fig 46*) but its concentrating function not completely destroyed, and it is accompanied by some



FIG 45—Intrinsic scar due to chronic fibrotic change similar to organized hour-glass in the stomach



FIG 46—Slight damage to mucosa shown by poor concentration, and numerous stones present in the fundus



FIG 47—Gall bladder slightly dilated, function subnormal. Several gall-stones are shown, one in the cystic duct acting as a ball-valve allowing the gall-bladder to fill, but not allowing it to empty



FIG 48—Pericholecystitis shown by adhesions to descending duodenum. Notice the absence of valvulae conniventes

fibrotic changes in the wall. If 40 per cent residue is present three hours after the administration of a fat meal, and the density is equal to that observed on the original films, we believe one is justified in a diagnosis of chronic cholecystitis.

When the gall-bladder fails to fill at any of the periods throughout the examination, while the probabilities are that the reason for this phenomenon is a well-marked cholecystitis, other lesions may be the cause, and the radiologist should make a diagnosis of mal-function, at the same time searching by the meal and enema for other lesions which may account for the non-filling. A stone situated in the hepatic, cystic, or common ducts may be the answer to the non-visualization (*Fig 47*), and if sufficient calcium is present it will undoubtedly show upon the films.

Often an acute duodenal ulcer or a gastric ulcer close to the pylorus will give a non-filled gall-bladder, which at operation and under the pathologist's microscope is normal. Whether this is due to some communication between the special sympathetic branches where the ulcers are present and the sympathetic branches with which the gall-bladder is so richly supplied, we are unable to state at the present time. An acute appendix will often also give us the same non-filling in a normal gall-bladder, and other acute abdominal lesions we have found occasionally set up the same reflex.

Malignancy of the gall-bladder is demonstrated only by complete non-filling, unless there is extension to the duodenum, and then the deformity is noted by the meal. It is possible that sufficient healthy mucosa could remain in carcinoma of the gall-bladder to concentrate the dye and show an actual deformity as in carcinoma of the stomach when the barium meal is used, but in our experience we have never filled a gall-bladder when this type of pathology is present and we have had to satisfy ourselves with a diagnosis of mal-function (*Fig 48*).

Non-visualization may occur for other reasons. An enlarged kidney, retroperitoneal tumour, the high fundus of the uterus in the later months of pregnancy, or a large intra-abdominal cyst may press upon the cystic duct and thus not allow a normal gall-bladder to be demonstrated. Carcinoma of the hepatic ducts and hepatitis may also be the cause of non-filling.

Hydrops of the gall-bladder (*Fig 49*) may easily be mistaken for the shadow caused by the right kidney, as the viscus in its enlargement assumes a shape resembling the renal contour. The soft film will, however, show the renal shadow separate from the markedly distended gall-bladder filled with the poorly concentrated dye.

Biliary calculi (*Figs 50-52*) have been classified according to their chemical composition. From an academic standpoint this is very desirable, but the task of the radiologist is much simplified and less confusion caused in the mind of the clinician if they are simply classified according to the calcium content present. Those rich in calcium are known as 'opaque' to the ray, and those wholly lacking or possessing only a slight amount of calcium, such as cholesterol stones, as 'non-opaque'. The last-mentioned type will be missed on the film very often unless a fair degree of concentration of the tetraiodophenolphthalein has been obtained. Fortunately with these types of stones a large percentage of the cases show slight damage to the mucosa, and the gall-bladder is seen well filled with the concentrated salt, the stones appearing as small dark shadows in the white gall-bladder. Papillomata of the gall-bladder may simulate these stones. The differential diagnosis



FIG 49—Hydrops of the gall-bladder. The markedly distended gall-bladder is shown pressing upon the barium-filled transverse colon



FIG 50—Cholesterol stones in deformed gall-bladder due to pericholecystitis, and cystic duct dilated from obstruction



FIG 51—Cholecystitis as shown by non-filling of the gall-bladder. Numerous faceted stones present



FIG 52—Pericholecystitis shown by deformed gall-bladder. Function of gall-bladder subnormal. Cholesterol stones are present

lies in the fact that on the various films stones will move about and assume different groupings in the fundus, body, or neck. This is particularly well seen during the period of concentration and contraction of the viscus. With papillomata the grouping will always be the same, only a closer approachment of the individual shadows is noted as the serial films are made. Another frequent source of error or confusion with this type of stone is gas in the second portion of the duodenum. The fenestration of the valvulæ conniventes duodeni simulates stones or papillomata very closely, but on precise observation of the various films these shadows will be noticed partially within and partially without the periphery of the filled gall-bladder, thus definitely locating them within the duodenum. A polypus of the gall-bladder may simulate a non-opaque stone, but rarely does a stone of this type attain the size of the usual polypus without sufficient calcium content present to show a well-marked periphery. The opaque stones may be well visualized in a gall-bladder still capable of carrying on its concentrating function, the varying amount of calcium content showing denser than the dye-impregnated bile. From this we see that the presence of either type of stone may be demonstrated in a gall-bladder apparently functioning normally.

For a considerable time we were of the opinion that a gall-bladder possessing stones but carrying on a normal function might well be treated by watchful waiting. However, considering the high incidence of carcinoma with an early history of gall-stones we now feel that the demonstration of cholelithiasis should be accepted as an indication for surgery.

When a gall-bladder shows no filling and shadows suggesting gall-stones are present, one must always remember that many other calcified deposits in this region may simulate on the film the presence of stone, and one must be on guard that an erroneous diagnosis of gall-stones is not added to the correct one of mal-function as shown by the non-filled gall-bladder. Renal calculi, calcification of Glisson's capsule, calcified lymph-glands, calcification of the suprarenal (*Fig 53*), calcified rib cartilages, calcareous degeneration of the right renal artery, pancreatic calculi, dermoid cysts (*Fig 54*), moles on the skin, faecal concretions in colonic diverticula, all cause shadows simulating gall-stones, and each must be carefully differentiated.

A small percentage of gall-stones with cholecystitis perforate, establishing fistulæ into the adjacent viscera. We have seen these tracts established into the pylorus, the duodenum, and the colon (*Fig 55*). Unless the barium meal and enema are added to the investigation, only a partially correct conclusion is reached by the demonstration of mal-function, and the surgeon encounters a much more difficult operation than would be the case had the radiologist carefully prepared his work beforehand. By the use of the meal or enema, the fistula is well seen and the gall-bladder filled with the barium. Occasionally after such a perforation has occurred into the colon the first clinical manifestation points towards colonic obstruction, for this reason the radiologist should be on the lookout for a large stone at the site of obstruction. Gall-stones may be found at any point within the colon, in one case referred as subacute appendicitis we observed a perforated gall-bladder, the stones had dropped into the cæcum, and at our examination we found three gall-stones lodged in the appendix.

Ulcers of the pylorus and duodenum (*Fig 56*) may perforate into the common duct, setting up cholecystic symptoms, but with the barium meal the common and



FIG 53—Cholecystitis demonstrated by non-filling. Calcareous shadow is due to calcification of the right suprarenal



FIG 54—Gall-bladder carrying on normal function, but a double fundus is seen. Shadow above the gall-bladder is due to a dermoid cyst. Notice the shadow of bone fragments and of teeth



FIG 55—Fistula established between gall-bladder and colon following ulceration. Gall-bladder filled during flow of enema



FIG 56—Perforated duodenal ulcer. Fistula established into common bile-duct. Notice common duct and hepatic ducts filled with barium

cystic ducts and the hepatic tree will be visualized, showing the primary lesion to be gastro-intestinal and that of the gall-bladder secondary

Frequently the first lead given towards gall-bladder disease is found in deformity of the pylorus and cap simulating either pyloric ulcer, early gastric carcinoma, or duodenal ulcer. However, this deformity is not as constant as in any of these conditions, and is caused by a veil or membrane, the so-called Morris membrane, extending from the gall-bladder and due to pericholecystitis. If the gall-bladder fills with the function test, deformity in its contour will be noted owing to these adhesions, and on fluoroscopic examination and palpation its mobility will be much lessened. In the normal stomach and duodenum during the screen examination and upon the films, the duodenal caput should ride true upon the pylorus, with the sphincter placed centrally, and angulation of the duodenal bulb or inconstant deformity should make one suspect the presence of pericholecystitis. Pericholecystic adhesions may also extend to the second portion of the duodenum. This part of the first loop of small intestines may be angulated, lacking in fenestration owing to tension from the adhesions, or present a convex curve due to pressure from the dilated gall-bladder. The adhesions may also extend to the colon, and in the proximal part of the transverse colon a pseudo-hepatic flexure is formed.

Pylorospasm is a frequent indication of gall-bladder disease during the fluoroscopic examination and upon the films. The pyloric canal—and by this we mean the portion occupied by the fan-muscle—never contracts or expands in a normal manner. We suspect, but at this time have not absolute proof, that the reason of this is some communication between the special branch of Opitz controlling the fan-muscle and the sympathetic supply of the gall-bladder.

THORIUM DIOXIDE IN GALL-BLADDER INVESTIGATION

When the dye has been concentrated in the gall-bladder and the position of the viscus is in marked variation with the habitus of the patient, it is probably due to some enlargement or displacement of the liver. Radiological examination by the ordinary technique will be of little assistance, but the administration of colloidal thorium dioxide intravenously will give a well-visualized liver, and a detailed study of structural changes may be made. Cirrhosis, both the hypertrophic and atrophic type, syphilis, hydatid cysts, polycystic disease (*Fig 57*), secondary carcinoma, and retroperitoneal tumours displacing the liver may all be shown. We have now used this solution for more than two years, and as yet have seen no ill effects, nor have any of our experimental animals killed from time to time shown any histological changes which could be interpreted as injurious to the liver or spleen, where 80 per cent of the retention of this medium occurs.

Not only as an indicator of gall-bladder function may the sodium tetraiodophenolphthalein be used, it is indeed valuable also as a basis for study of liver function, similar to the tests of Rosenthal with phenoltetrachlorophthalein. We believe that patients with a fairly definite gall-bladder history showing complete retention of the dye will be much better surgical risks if, instead of being submitted to immediate operation, they are placed at rest in bed on an abundant carbohydrate diet in the form of glucose, some calcium, and large amounts of

water, and re-examined radiologically at a later period. In these cases we have found at the second examination concentration of the dye present, the complete retention having passed, and surgery may be undertaken with the expectation of a lower fatality rate.

Occasionally cases of intestinal allergy will give symptoms identical with those of gall-bladder disease. If these cases are investigated with cholecystography and



FIG 57—Polycystic disease of the liver shown by thonium dioxide. This case was referred as probable carcinomatous secondaries in the liver.

the ordinary opaque meal and found normal, we would suggest a series of sensitization tests before a definite medical or surgical regime is established.

In conclusion, may we plead for the closest collaboration between the physician, the surgeon, the pathologist, and the radiologist? If this is brought about, we shall not only increase the number of correct diagnoses, but also lower the mortality rate from disease in the upper right quadrant.

CHANGES IN THE BLOOD-SUGAR LEVEL ASSOCIATED WITH SURGICAL OPERATIONS

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THE work which follows was done in the Surgical Professorial Unit of St Bartholomew's Hospital at the instigation of its Director, Professor Gask

It is a fairly general routine procedure to administer glucose by the rectum to patients who have undergone a severe surgical operation. Recently doubt has been thrown on the wisdom and usefulness of this procedure by Levi¹. His observations led him to conclude that glucose rectal salines are not only useless but even harmful. Indeed, he proposed that in cases of post-operative shock it would be more in the interests of the patient to administer insulin.

The level of the circulating blood-sugar is a balance between income and expenditure. Ingested carbohydrates are absorbed from the alimentary tract in the form of glucose and then carried to the liver, where they are converted into, and stored as, glycogen. As required, this glycogen is reconverted into glucose and passes into the blood-stream, maintaining the blood-sugar level within certain limits, i.e., 80 to 120 mgrm per cent. This represents a physiological cycle: Portal sugar \rightarrow liver glycogen \rightarrow circulating sugar. This cycle is influenced by nervous-hormonic factors. The sympathetic-adrenalin mechanism promotes the change of glycogen into sugar, the vagal-insulin the conversion of sugar to glycogen and perhaps the utilization of sugar by the tissues. Anything tending to augment the sympathetic-adrenalin mechanism, such as activity, an increased metabolic rate as found in thyreotoxæmia, emotion, anæsthesia, restlessness, pain, or trauma, produces more adrenalin, thereby raising the level of the blood-sugar. On the other hand, food, rest, sleep, tranquillity, and freedom from pain augment the vagal-insulin mechanism, tending to produce a lowering of the level of the blood-sugar. Thus from general principles we should expect that emotionally stable patients, who are in good condition before the operation and whose convalescence is uninterrupted by pain or other adverse factors, should have a lower blood-sugar level than patients in whom these conditions are reversed. In other words, we should expect the level of the blood-sugar to run parallel with the clinical condition of the patient.

In view of the above criticism of the post-operative treatment of surgical cases it is of importance that the changes in the blood-sugar level as affected by surgical operations should be re-investigated. In the course of such an investigation it is necessary to consider first of all the effect of the anæsthetic. It is well known that the amount of sugar in the blood rises during anæsthesia. That this is due to the liberation of adrenalin is known, and recently Reid and Banerji² have shown that the actual sugar level in animals depends quantitatively on the amount of adrenal medulla available. In addition to the influence of the anæsthetic it is necessary to estimate if possible the extent, duration, and variation in the blood-sugar level which

may occur as the result of the operation itself. With such knowledge accurately obtained it might then be possible to assess the need or otherwise for glucose rectal injections and perhaps judge if other procedures might be useful.

METHOD OF INVESTIGATION

The present investigation was confined to patients undergoing major operations. In order that the results obtained from all patients might be as parallel as possible, the blood was collected at the following periods: (1) On the day previous to the operation after a fast of twelve hours—this gave a resting level, (2) Half an hour before the operation, (3) After the induction of surgical anaesthesia when the skin incision was being made, (4) At various stages throughout the operation, these corresponding as far as possible in all cases, (5) Immediately after the operation when the patient had been returned to bed, (6) Six hours after this, (7) On the following morning, approximately twenty-four hours after the operation.

Blood was obtained from the finger except during the operation, when it was taken from the great toe because this was less disturbing to those taking part in the operation. The same puncture usually sufficed for all the specimens collected during the operation. The time at which the samples were taken was accurately noted for the sake of producing graphs (*Figs 58-66*) which could be easily compared.

The estimation of the blood-sugar was by a modified Hagedorn and Jensen's method as used in this hospital by Dr G. Graham.³ By this method only small quantities of glutathione, the other main reducing substance in the blood, are included in the estimation. We observed the following special precautions in making the estimations. The blood was accurately measured in a narrow-bore pipette designed to deliver 0.1 c.c. All filter papers were, of course, free from starch. We avoided handling these as far as possible, since this is a known way of contaminating them with reducing substances. The papers were wetted with distilled water before filtering the precipitated proteins to prevent the loss of glucose during washing. We found that extreme care was necessary in the measurement of the known amount of potassium ferricyanide, an error of one drop affecting the reading to the extent of 10 mgrm per cent. The titrations against thio-sulphate were done with a microburette in order to obtain an accurate end point. Two samples of blood were collected for each estimation, the mean between the two being taken as the reading. Ordinarily a difference of 10 mgrm per cent between the two estimations is considered good. By strict attention to the details of the technique the differences in our readings were within 5 mgrm per cent, and in most cases not more than 1 or 2 mgrm per cent. All results with a difference of above 5 mgrm per cent were discarded. The thio-sulphate solution was freshly made for each series of estimations.

CONCLUSIONS DRAWN FROM THE GRAPHS

Pre-operative Observations—Examination of the curves obtained shows that the blood-sugar level in patients about to undergo a major operation does not vary much from the resting level. It is known that persons who are in a state of anxiety or apprehension have a raised blood-sugar level. It might perhaps be

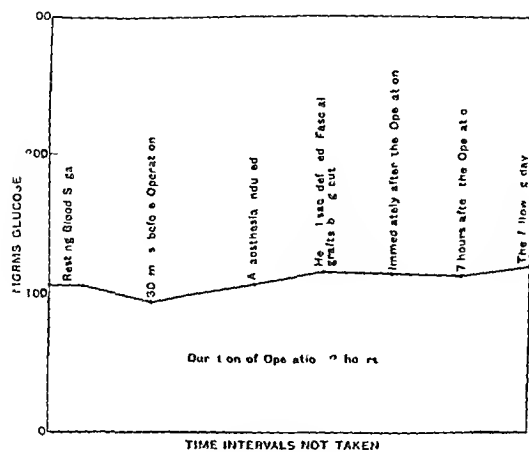


FIG 58

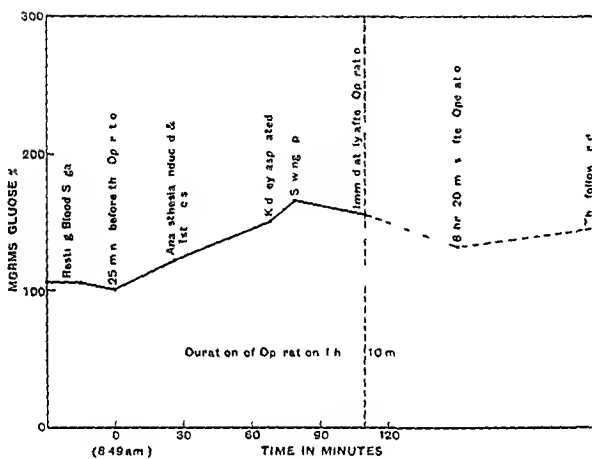


FIG 59

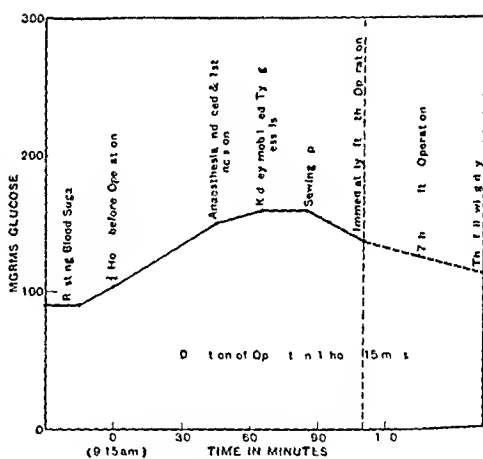


FIG 60

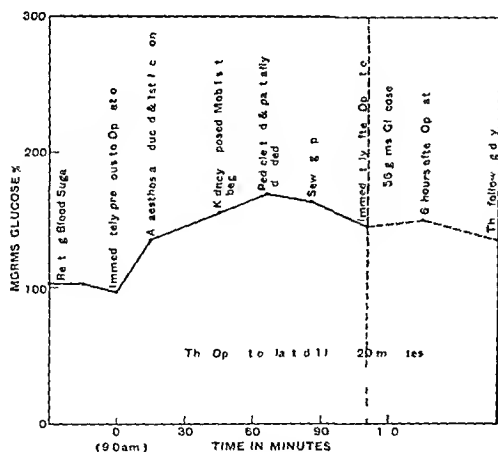


FIG 61

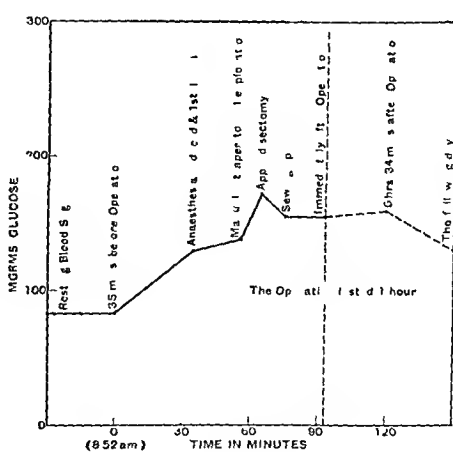


FIG 62

BLOOD-SUGAR LEVEL IN SURGERY

83

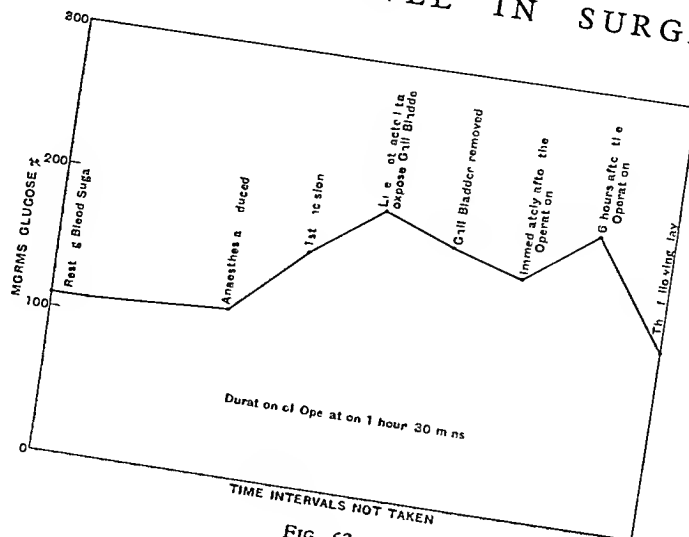


FIG 63

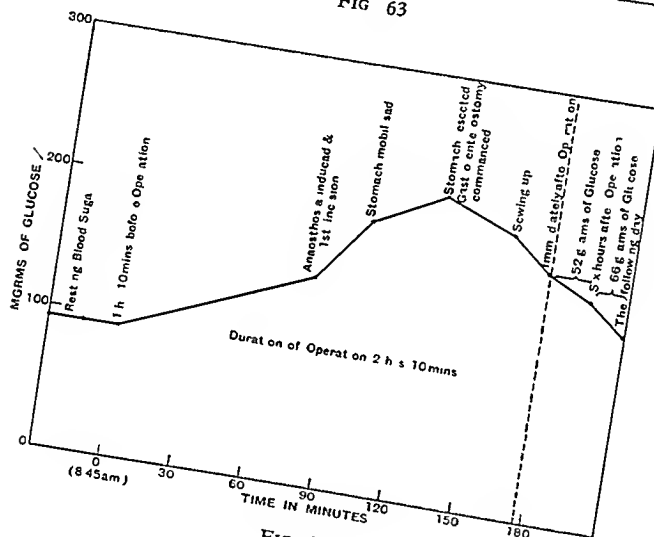


FIG 64

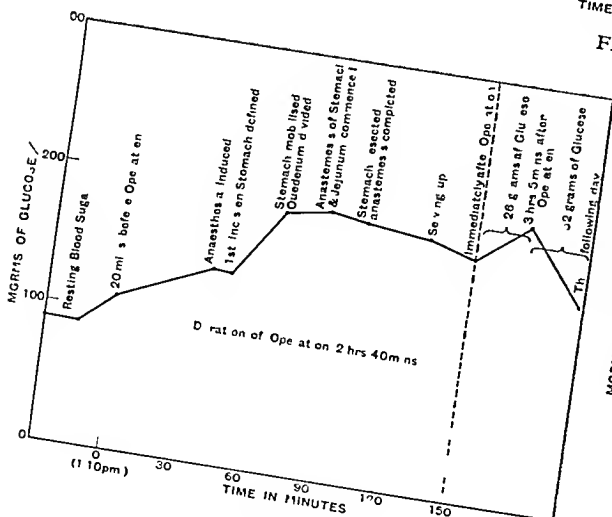


FIG 65

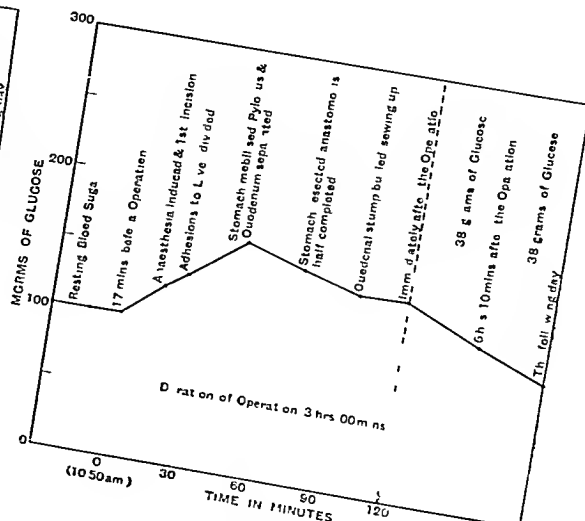


FIG 66

surmised from this that the ordinary hospital patient is not apprehensive or even greatly concerned about his approaching operation. We do not know whether this is a tribute to surgical skill or to the mental capacity of the patients.

Effect of Anæsthesia—Induction of surgical anæsthesia has produced an increase in the blood-sugar level of variable extent. The average lies between 20 and 50 mgrm per cent. The anæsthetic used in all cases was gas, oxygen, and ether. Induction of anæsthesia caused a larger average rise in males (36.5 mgrm per cent) than in females (23 mgrm per cent). The number of female cases was, however, only three. This suggests, so far as it goes, that the threshold of adrenalin release may be higher in females than in males. Perhaps this is another indication of the greater degree of stoicism usually attributed to women.

Further comparison of the graphs brings out the fact that the maximum height of the sugar level above its resting value is not correlated with the duration of the anæsthesia.

Table I—RELATION BETWEEN DURATION OF OPERATION AND RISE OF BLOOD-SUGAR LEVEL

MRGM PER CENT ABOVE RESTING LEVEL	DURATION
124	2 hr 10 min
101	2 hr 40 min
(a) 88	1 hr 00 min
88	1 hr 30 min
70	1 hr 15 min
67	1 hr 20 min
(b) 63	3 hr 00 min
62	1 hr 10 min
10	2 hr 00 min

The relationship between the maximum rise and the duration of the operation is set out in Table I. Taking the two extreme cases (a and b) to illustrate this point, it will be seen that in the operation lasting one hour there was a rise of 88 mgrm per cent while in the other lasting three hours there was only a rise of 63 mgrm per cent.

Nature of the Operation—The nine cases investigated can be divided into two groups: (1) Intraperitoneal, and (2) Extraperitoneal. In Group 1 (3 partial gastrectomies, 1 cholecystectomy, and 1 exploratory laparotomy followed by appendicectomy) the average maximum rise was 93 mgrm per cent, while in Group 2 (2 nephrectomies, 1 nephrostomy, and 1 ventral hernia repaired with fascial grafts) the average maximum rise was 52 mgrm per cent. From this it seems that operations which take place nearer to the splanchnic area, i.e., in the vicinity of the solar plexus, cause a greater rise in the blood-sugar level than those placed at a distance from this point. It is interesting to note in Fig 63 that the maximum blood-sugar level was reached while the liver was being palpated. This relation is even better shown in Fig 62, where, ten minutes after the manual exploration of the abdominal viscera, there was a peak in the blood-sugar level.

Post-operative Observations—In patients who appeared comfortable and relatively free from pain on the following day, the blood-sugar was found to have

reached a lower level than in those who were uncomfortable and in pain. This is shown in *Table II*, where short clinical notes and the chief points of moment in the graphs are tabulated.

Four patients were given glucose rectal salines, which were retained, and the blood-sugar level was estimated on the following day. Of these four cases the blood-sugar level had already returned to normal in one case and in the other three it was falling. It was therefore concluded that these patients were utilizing the glucose administered by the enemata.

DISCUSSION

From the evidence produced it appears that the hypothesis made earlier, that the blood-sugar level runs parallel with the clinical condition of the patient, is justified. Operations on the upper abdomen around the coeliac ganglia are more marked in their effect on the blood-sugar level than those placed more remote from this region, irrespective of their duration. If a high blood-sugar level is to be avoided gentle handling is an essential factor in this neighbourhood.

It has been claimed that in a series of normal and diabetic patients the administration of 50 to 80 gm of glucose in 500 c.c. of saline occasioned either a fall or at the most only a slight rise in the blood-sugar level. It was concluded therefore that sugar was not absorbed from the colon, in contrast to its absorption from the stomach, this leading to a pronounced change in the blood-sugar level. In our series the enemata were always retained and the bowels remained unmoved for twenty-four hours. So it seems reasonable to us to conclude that absorption had taken place. The effect on the blood-sugar level will depend on several factors, the rate of absorption, the rate at which the sugar is being produced or used up. The problem of absorption cannot be resolved by direct appeal to the blood-sugar level at any one moment. Absorption of glucose from the rectum is at the rate of 6 gm per hour (Starling) or 3 gm per half hour. 50 gm of glucose given by mouth produces a maximum rise of about 60 mgrm per cent in the blood-sugar level in half an hour. Assuming all factors to be constant, 3 gm would only give a rise of 4 mgrm per cent. Thus no great rise can possibly be expected to occur when glucose is given per rectum. Further detailed work on this problem has been done by Collens and Boas,⁴ who produced additional evidence for the absorption of glucose from the rectum. In all our cases the blood-sugar level, although above normal, continued, after no rise or a very slight one, to fall despite glucose enemata. From our observations we have no evidence that glucose enemata are harmful or irritant. Rather it would seem that their use is justified on the ground that they replace the depleted sugar stores of the body.

The raised blood-sugar level shown in these curves must depend to some extent upon a diminished utilization of glucose by the tissues, since the patients are in a depressed condition during and for some time after an operation. It has been suggested that insulin would be of value presuming that it would increase utilization. The carbohydrate stores must have been depleted to some extent during the operation, and an injection of insulin would still further diminish these stores unless it was combined with glucose. In any case, if the area beneath a graph made from the curve in *Fig 64* (extended to cover a twenty-four hour period) is calculated, the amount of glucose represented is 27 gm, which is

Table II—CHANGES IN THE BLOOD-SUGAR LEVEL ASSOCIATED WITH SURGICAL OPERATIONS
(NINE CASES)

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Table II—CHANGES IN THE BLOOD-SUGAR LEVEL ASSOCIATED WITH
(NINE CASES)

CASE No	AGE AND SEX	OPERATION	DURATION	GLUCOSE IN MGRM PER CENT						RECTAL GLUCOSE	CONDITION		
				Resting Blood-sugar	Pre operative Rise or Fall	Rise after Induction of Anaesthesia	Maximum Rise above Resting Level	B S Twenty-four hours after Operation			Pre-operative	Post operative	
1	M 61	Hernia	Hrs 2 Mins 00	105	-11	1	10	16	Grm 0	Good	Good	Little pain	
2	F 28	Nephrostomy	1 10	105	-6	16	62	40	0	Pyrexial	Some pain	Some pain	
3	M 40	Nephrectomy	1 15	90	+13	60	70	22	0	Fair	Fair	Little pain	
4	M 62	Nephrectomy	1 20	102	-5	33	67	33	56	Fair	Fair	Cough	
5	F 28	Laparotomy	1 00	83	0	47	88	47	0	Good	Good	Vomiting	
6	F 40	Cholecystectomy	1 30	110	—	6	88	20	118	Fair	Fair	Good	
7	M 46	Partial gastrectomy	2 10	93	0	53	124	47	78	Hb per cent low	Hb per cent low	Some pain	
8	M 64	Partial gastrectomy	2 40	89	+21	50	101	61*	0	Good	Good	Weak and feeble	
9	M 49	Partial gastrectomy	3 00	103	0	22	63	0	76	Good	Good	Very good	

* Eighteen hours after operation in this case

* Eighteen hours after operation in this case

equivalent to 108 calories. The basal metabolic requirements may be taken as 1800 calories. Thus it will be seen that the amount of excess of sugar circulating in the blood is small. The case on which this calculation is based showed the highest maximum rise in blood-sugar level. Now in *Fig 58* this area is negligible, so that it will be seen that the routine administration of insulin and glucose as a part of post-operative treatment cannot be expected to have any specific beneficial effect.

From the cases which are the basis of this report we are inclined to believe that a persistently high blood-sugar level is a feature to be expected when the patient is not doing well. Such an expectation, if confirmed, would perhaps give some point to the suggestion that insulin combined with glucose might be of some service.

SUMMARY

- 1 Pre-operative anxiety, as measured by the change in level of the blood-sugar, must be very slight.
- 2 The duration of the anæsthesia does not determine the height of the maximum rise.
- 3 There is a rise in the blood-sugar level during major operations, particularly marked when the splanchnic area is involved.
- 4 The rise in the amount of the blood-sugar level depends far more on the nature of the operation than on its duration.
- 5 We find no evidence that glucose rectal salines as a routine post-operative procedure have any specific effect. There is no reason to suppose that they are either useless or harmful, and they may be of value in that they form a convenient method of post-operative feeding.
- 6 The use of glucose and insulin in post-operative treatment is discussed.

REFERENCES

- ¹ LEVI, D, "A Note on the Glucose Enema and its Value in Post-operative Treatment" *Brit Jour Surg*, 1927, xv, 282.
- ² REID, C, and BANERJI, H, "The Adrenals and Anæsthetic Hyperglycæmia", *Jour of Physiol*, 1933, lxxviii, No 4, 370.
- ³ GRAHAM, G, *The Pathology and Treatment of Diabetes Mellitus*, 2nd ed 1926 206.
- ⁴ COLLENS, W S, and BOAS, L C, "Absorption of Dextrose by Rectum" *Arch Internal Med*, 1933, lxi 317.
- ⁵ STARLING, E H, *Principles of Human Physiology*, 1912, 815.

DIVERTICULA OF THE VERMIFORM APPENDIX*

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THE first attempt at a comprehensive survey of diverticula of the vermiform appendix appears to be that of Herb in 1907. Further papers appeared in 1911 (MacCarty and McGrath), 1919 (Moschowitz), 1921 (Wilkie), 1923 (Stout), 1927 (Chase), and 1928 (Pack and Scharnagel).

During the five-year period 1927-31, 1493 appendices have been removed at operation at King's College Hospital. Of these, 8 showed the presence of diverticula. The percentage incidence represented by these figures is, therefore, 0.53 per cent. These figures, however, must almost certainly exaggerate the incidence, for in quite a large number of cases the normal appendix has been removed when the abdomen was opened for some other surgical condition and the fact not made sufficiently prominent in the records to enable one to trace it. The total number of appendicectomies therefore exceeds the above figure. Even when an allowance is made for this fact, the incidence is undoubtedly higher than the scant reference to the subject in the literature leads one to imagine. No single case has been observed in the last 2680 consecutive post-mortems. Diverticula of the appendix are easily seen, and it is unlikely that the examiner would miss them if present. The explanation of this striking fact is probably that diverticula of the appendix are almost invariably associated with inflammation of the appendix, and this leads to operative removal.

In none of 4631 barium meals and 695 barium enemas were diverticula of the appendix revealed by X rays. For the two cases of diverticulosis of the appendix diagnosed by X rays the author is indebted to colleagues. These cases are of considerable interest, for the appendix shares in a general diverticulosis of the whole colon. As will be seen, in the majority of cases in the author's pathological specimens there is an obstruction to the lumen of the appendix near the cæcal end. Such an obstruction will prevent the barium from entering the appendix, so that X rays will fail to reveal the diverticula. Thus in diverticulosis of the appendix we find an exception to the general rule that the diagnosis of intestinal diverticula depends upon X-ray examination.

The only figures of incidence which the literature offers are those of MacCarty and McGrath, who found 17 cases of diverticula in 5000 specimens (0.34 per cent), and those of Moschowitz, who found 4 cases in 1500 examinations (0.26 per cent).

Age and Sex Incidence—Diverticulosis of the appendix shows no parallel in respect of age incidence with diverticula elsewhere in the intestine. The author's 12 cases (including the X-ray cases) show a wide range, the youngest

*A section of the Jacksonian Prize Essay, 1932

being 18 and the oldest 64. The average age is only 42. Six were men and 6 women. The cases are as follows —

SPECIMEN	CASE No	AGE	SEX	DURATION OF SYMPTOMS
1	198	18	F	48 hours
2	199	64	M	2 months
3	200	41	F	12 months
4	201	48	M	7 years
5	202	27	M	2 years
6	203	30	F	48 hours (?)
7	204	28	F	48 hours
8	205	21	M	2 years
9	206	48	F	6 years
10	207	55	M	5 years
11*	168	58	M	No symptoms due to appendix
12*	209	62	F	No symptoms due to appendix

* Diagnosed by X rays

In MacCarty and McGrath's series of 17 cases, by a curious coincidence, the youngest and oldest patients were the same ages as the author's, viz., 18 years and 64 years respectively. The average age at operation was 34 years, and the average duration of symptoms 7 years.

DESCRIPTION OF SPECIMENS

In all cases the diverticula were multiple. Three were associated with acute, and seven with chronic, appendicitis.

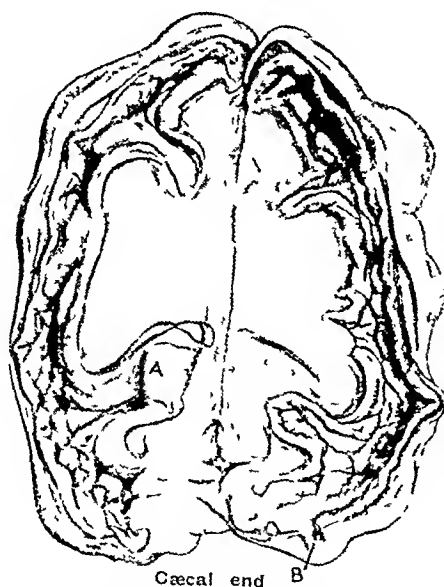


FIG 67 — Case 198. Diverticulitis of the appendix.
(Miss Barclay-Smith)

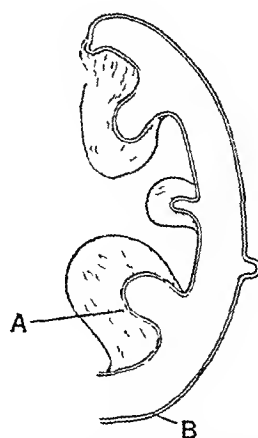


FIG 68 — Case 198. Diagrammatic representation of the specimen illustrated in Fig 67. (Miss Barclay-Smith)

Specimen 1 (Case 198) — The specimen (Fig 67) was removed at operation from a girl of 18 who suffered from symptoms of acute appendicitis. There was

no previous history of pain. It shows an enlarged appendix cut through from the convex border, and opened out. The incision has also divided the meso-appendix. The whole of the appendix shows evidence of severe acute inflammation, being almost gangrenous in places. The wall is greatly thickened, and the mucous membrane dark from extreme congestion. Microscopic section shows an extensive infiltration of all the coats of the appendix with blood. The mucous membrane preserves the normal rugose appearance, and there is thus no evidence of persistent high pressure inside the lumen. The meso-appendix is thickened to many times the normal size. Pushing into it from the concave aspect of the appendix are four pouches, which have been bisected. The first and third of these (starting from the cæcal end) are of very large size. The second can only be seen on one half of the specimen, and the fourth is small, and near the tip. The line of section passes through a fifth diverticulum, situated on the convex border, $\frac{1}{10}$ in from the cæcal end.

The first, second, and third diverticula form large, firm, rounded swellings, bulging into the meso-appendix, and can be clearly seen on the reverse side of the specimen. There is a cleft between the first and second pouches (*Fig 67*).

The wall of each of the first three diverticula is very thick, that of the first one measuring fully $\frac{1}{2}$ in across. The position and relative thickness of the diverticula are best seen in the diagrammatic representation in *Fig 68*. There is no obstruction to the opening from the appendix into the cæcum.

A section for microscopic examination was taken from A and B. A shows the wall of the first diverticulum and B a portion of the wall of the appendix itself. The whole of the diverticular wall is infiltrated with blood, a condition of hæmorrhagic infarction. The blood-cells lie in a framework of œdematous fibrous tissue, which represents the altered submucous coat. No muscle tissue is seen in the wall of the diverticulum, but is present in the section of the appendix wall, lying beneath a markedly thickened submucosa, which is infiltrated with blood.

SUMMARY—An acutely inflamed appendix, showing hæmorrhagic infarction, bears five diverticula. Four pass into the meso-appendix and one from the convex surface. The wall of the diverticula contains no muscle.

Specimen 2 (Case 199)—The specimen (*Fig 69*) was removed at operation from a male patient aged 64 years, who had suffered from symptoms of chronic appendicitis for two months. The specimen has been divided along its long axis and opened to display the lumen. The appendix is shorter than the usual, and $\frac{1}{2}$ in from the tip is kinked to a right angle. There is a deposit of fat in the meso-appendix at the angle thus formed. The lumen distal to the

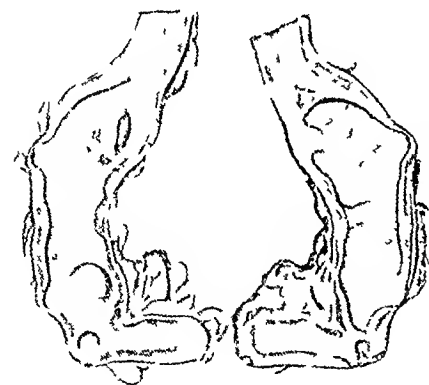


FIG 69—*Case 199*. Diverticula of the appendix. The cæcal end of the lumen is obstructed, and the distal third obliterated (*Miss Barclay Smith*).

kink is completely obliterated by fibrous tissue, and the lumen at the cæcal end is markedly stenosed. Between these two obliterated portions, the lumen of the appendix is widely distended, and its wall is comparatively thin. The mucous

membrane is smooth. Opening out from this distended portion are some small diverticula. These are wide-mouthed, and have an extremely thin wall. If held up to the light, the wall looks like thin tissue paper, having no muscular coat. The fate of the muscle coat is clearly shown on naked-eye examination. If the convex margin be traced from the cæcal end to the tip, a small wide pouch is seen. At the mouth of this pouch there is a wide gap in the muscle coat. This occurrence is also shown an inch further along the border. Except for the gaps, the muscle coat of the ballooned part of the appendix is well developed, and shows a definite hypertrophy near the centre of this portion. Apart from the obliterative process at either end of the appendix, there is little macroscopic evidence of chronic inflammation. The diverticula themselves are quite free.

No microscopic section of this specimen was made. One needs to take a section completely across the appendix and a diverticulum to be of value, and such, unfortunately, would destroy the specimen. Other specimens (Nos 9 and 10) were sacrificed to this end.

SUMMARY—In this case the lumen of the appendix is stenosed at both ends. As a result of the proximal obstruction, the middle part is ballooned out. The wall is thin, but the muscle coat is slightly hypertrophied. There are gaps in it, through which the mucous membrane has herniated to form diverticula. These have very thin walls. The situation of the diverticula is not regular. There is one on the antimesenteric aspect, and the two largest are situated on the surface between the concave and convex aspects.

Specimen 3 (Case 200)—The specimen (Fig 70) was removed at operation from a woman aged 41 who had suffered from attacks of pain in the right side of the abdomen for a little over twelve months. The specimen has been split longitudinally from the convex surface, and shows a long and thickened appendix. The lumen near the cæcal end is very narrow, but not completely obstructed. A further narrowing occurs about half way along. There is no distension of the lumen in any situation. The mucous membrane is very much thickened throughout, and the lumen is not easily traced. Small bristles have been placed in the mouths of pockets formed by the mucous membrane pushing through the muscle coat. Most of these are small, but on the convex aspect, just beyond half way, there is a flask-shaped protrusion. The mucous membrane lining this is of the same thickness as in the rest of the appendix. This diverticulum is empty and collapsed—not ballooned out as in *Specimen 2* (see Fig 69). In fact, there is no ballooning of any of the small pockets in this specimen.

The submucous coat and peritoneal coat are both irregularly thickened. Chief interest lies in the muscularis, which can easily be traced on naked-eye examination.



FIG 70—Case 200. Diverticula of the appendix. The muscular coat is well defined, except in the proximal half on the convex aspect. At A a portion has been removed for section.

Near the cæcal end it is well developed, and looks thicker than in the normal appendix (cf *Fig 88*) Near the narrowing about the centre of the specimen,



FIG 71—*Case 200* The microscopic appearance of the wall at A, in the specimen illustrated in *Fig 70*. The muscle coat is partly disorganized by chronic inflammatory changes. A, Submucosa, B, Muscle (*Mr S Steward*)

SUMMARY—In this specimen there is chronic widespread inflammation of the wall of the appendix, with a hypertrophied muscle in which gaps are present. Through these gaps the mucous membrane has become everted to form small diverticula. The gaps in the muscle coat are probably the result of fibrosis. There is no distension of either the lumen of the appendix or of the diverticula.

Specimen 4 (*Case 201*)—The specimen (*Fig 72*) was removed at operation from a man aged 48 who had suffered attacks of pain for seven years. The specimen has been split along the convex border, and opened out to display the lumen. The appendix is very thick and stumpy. The muscle coat is abnormally well developed, and is interrupted at intervals by septa of fibrous

there is a considerable local hypertrophy on the convex aspect, with a deficiency opposite. On the mesenteric (concave) border, the muscle coat continues without interruption until the tip is reached. On the convex aspect, however, there is an abrupt end through which the pouch described above passes. On the other side of this pouch the muscle reappears, but becomes practically lost soon after, only a thin irregular line being traceable. In this situation the mucous membrane shows many small pouches, passing out under the serous coat. Near the tip a wedge has been cut for microscopic examination (*Fig 71*). The mucous membrane shows three very deep crypts, passing deeply into the submucosa, which is very much thickened from fibrosis. The muscle coat showing in the illustration is thick, but very irregular. There is no sharp distinction between it and the submucous coat, and there is a considerable fibrosis around the groups of fibres. The longitudinal coat disappears entirely at one aspect, and a little beyond this, near the crypts of mucous membrane, the circular coat also ceases.



FIG 72—*Case 201* Diverticula of the appendix. The lumen is tortuous and contains faecal material (*Mr A Edmunds*)

tissue Half-way along the antimesenteric margin this fibrous interval has expanded under the peritoneal coat to form a small spherical knob of tissue, which is slightly hollowed out This hollow communicates by a narrow channel with the interior of the appendix, so that the knob is actually a diverticulum

The mucous membrane has been thrown into folds so that the lumen, which cannot be traced, crosses and recrosses the mid-line, and has a honeycombed appearance This appearance is most marked in the middle of the specimen, opposite the small knob-like diverticulum In this portion the lumen is filled with darkly stained faecal material

SUMMARY—In this case the most marked features are the folds produced in the mucous membrane by shortening of the other coats of the intestine—especially the muscle coat The latter is thickened, but is interrupted by fibrous septa, at one of which sites a hernial diverticulum has occurred

Specimen 5 (Case 202)—The specimen (*Fig 73*) was removed at operation from a man of 27 who had had symptoms of chronic appendicitis for about two years It consists of one-half of the appendix divided in the long axis in the line of the meso-appendix, and a segment of ileum, to which the tip of the appendix was so firmly adherent that resection was necessary

Apart from some thickening of all coats, the proximal third of the appendix is not grossly deformed The lumen, except for the first $\frac{1}{3}$ in, is, however, narrowed almost to the point of obliteration In the distal two-thirds the mucous membrane is thrown into folds The spaces between the folds are dilated to a flask shape These have a narrow communication with the lumen, except at the distal end of the appendix, where the distension is more marked, and the flask-shaped dilatations freely intercommunicate At the junction of the proximal third and distal two-thirds, the lumen is carried right across the mid-line and passes into a small diverticulum, which has penetrated through the muscle coat The flask-shaped pouches of the mucous membrane do not penetrate completely through the muscle coat except at one point along the convex border, $\frac{1}{3}$ in from the tip The muscularis in contact with the pouches, however, is extremely thin, and it is evident that had the condition been permitted to go on, they would have herniated completely through the muscle coat, owing to pressure atrophy of the latter, and complete diverticula would have resulted

SUMMARY—In this case there is chronic inflammation chiefly marked in the proximal third of the appendix, with partial or complete obstruction to the lumen Distal to this, the mucous membrane is thrown into folds, and the lumen distended so that a series of pouches are formed The distension is most marked at the tip

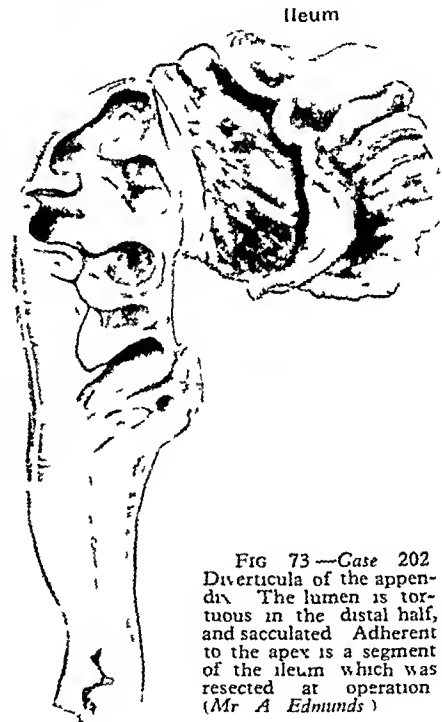


FIG 73—Case 202
Diverticula of the appendix The lumen is tortuous in the distal half, and sacculated Adherent to the apex is a segment of the ileum which was resected at operation (Mr A Edmunds)

of the appendix At the junction of the proximal third and distal two-thirds, the lumen has been carried towards the concave aspect, and communicates with a small hernial diverticulum

Specimen 6 (Case 203)—The specimen (*Fig 74*) was removed at operation from a woman of 30 who had symptoms of acute appendicitis There had been no symptoms previous to this attack The specimen consists of one half of the appendix split in the long axis in the line of its mesentery The proximal portion is acutely inflamed, especially at one point where there is a perforation, surrounded by an area of gangrene The meso-appendix is thickened from œdema The lumen of the proximal end is completely obliterated, and in the distal half is distended Four well-formed hernial diverticula open from it by wide mouths Two of these are on the meso-appendix aspect, and two on the convex margin The former two are acutely inflamed In the intervals between these diverticula, the mucous membrane dips down into the thickened submucous coat, in some cases as far as the muscularis These deep crypts are probably diverticula in embryo The muscular coat, though pale from œdema, is very much thicker than usual except at the tip It is interrupted at intervals by the diverticula, and also by the perforation

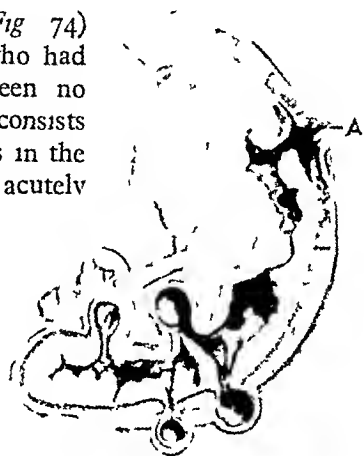


FIG 74—Case 203 Diverticula of the appendix The muscular coat between the diverticula is hypertrophied Perforation (A) has occurred near the cœcal end (r through a diverticulum)

It is impossible to say whether there was originally a diverticulum at the site of perforation Although there is no history of previous symptoms, the hypertrophy of the muscularis, the thickness of the submucous coat, and the stenosis of the lumen near the proximal aspect all suggest that there had been previous chronic inflammation

SUMMARY—An acute appendicitis, in which perforation has occurred, is associated with well-marked hernial diverticula The proximal part of the lumen is obstructed, and the distal part distended The muscularis is hypertrophied and there has almost certainly been inflammation of long standing present

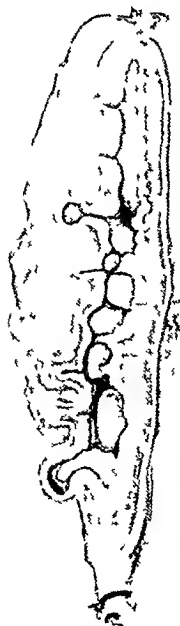


FIG 75—Case 204 Diverticula of the appendix Near the cœcal end the whole lumen is carried into a diverticulum These are all situated on the mesenteric aspect The lumen contains several concretions (Mr A Edmunds)

Specimen 7 (Case 204)—The hemi-section of an appendix (*Fig 75*) was prepared from a specimen removed at operation from a woman of 28 who had typical symptoms of acute appendicitis The proximal half of the appendix is acutely inflamed, and the meso-appendix is thickened from œdema The proximal part of the lumen is narrow but not obliterated, and just beyond this point the whole lumen is carried from the mid-line through the muscle coat to form a diverticulum

Several diverticula, varying from a wedge-shaped crypt of the mucosa to a fully formed flask-shaped pouch, pass through the muscle coat into the meso-appendix. These are *all* on the concave aspect of the appendix. The muscle coat is somewhat hypertrophied, especially between the diverticula. There is no interruption of its fibres on the side away from the meso-appendix. Six concretions lie in the lumen of the appendix, but the diverticula are empty. The submucous coat is not thickened and there is no evidence of previous gross inflammatory changes.

SUMMARY—An acutely inflamed appendix is associated with the presence of many diverticula, herniated through the muscular coat on the side of the meso-appendix. The *whole lumen* is carried into the first diverticulum, thus producing obstruction distal to it, so that concretions are present in the lumen. There has probably not been any gross inflammation of the appendix previous to the acute attack.

Specimen 8 (Case 205)—This specimen (*Figs 76, 77*) was removed at operation from a man of 21 who for two years had suffered from symptoms of chronic appendicitis. A giant congenital diverticulum was removed from this patient. Unhappily he developed ileus after the second operation, which was difficult owing to the position and fixity of the appendix, and died.

Fig 76 shows one-half of the appendix divided longitudinally, and *Fig 77* the distal portion of the other half, cut across the long axis. The remaining material was utilized for microscopic investigation.



FIG 76—Case 205. Diverticula of the appendix. The wall is thickened, and the lumen distended into sacculi. (Mr G B Davies)



FIG 77—Case 205. A section through the specimen shown in *Fig 76* near the distal end, and passing through a large diverticulum. (Actual size)

The whole appendix is greatly thickened and somewhat kinked. The meso-appendix is loaded with fat and bunched up into the angle formed by the kink. The lumen of the proximal half is normal. Opposite the bend in the appendix it disappears from view. This is probably because it has migrated to one or other side of the mid-line, as in some of the previous specimens. As the angle is passed, the lumen reappears as a small pocket to the left of the mid-line, but within the muscle coat. Distal to this, it forms three large pockets, separated by thick ridges of fibrous tissue. Even after serial sections of the opposite half of the appendix in the region of the pockets, it is difficult to trace the lumen between these dilations. On the half cut away there is a large diverticulum, shown in transverse section in *Fig 77*.

Except for an area on the concave border opposite the kink, the muscular coat is thickened and prominent. In the former situation it appears to be infiltrated with fat and fibrous tissue, and cannot be made out.

The diverticulum is situated on the concave border, just anterior to the meso-appendix. *Fig 78* is a low-power drawing of a section through the diverticulum and the adjacent wall of the appendix.

The diverticulum has no trace of muscle-fibres in its wall, with the exception of a muscularis mucosæ to be seen on high magnification. The submucous coat consists of loosely woven fibrous tissue, in which a large blood-vessel runs a

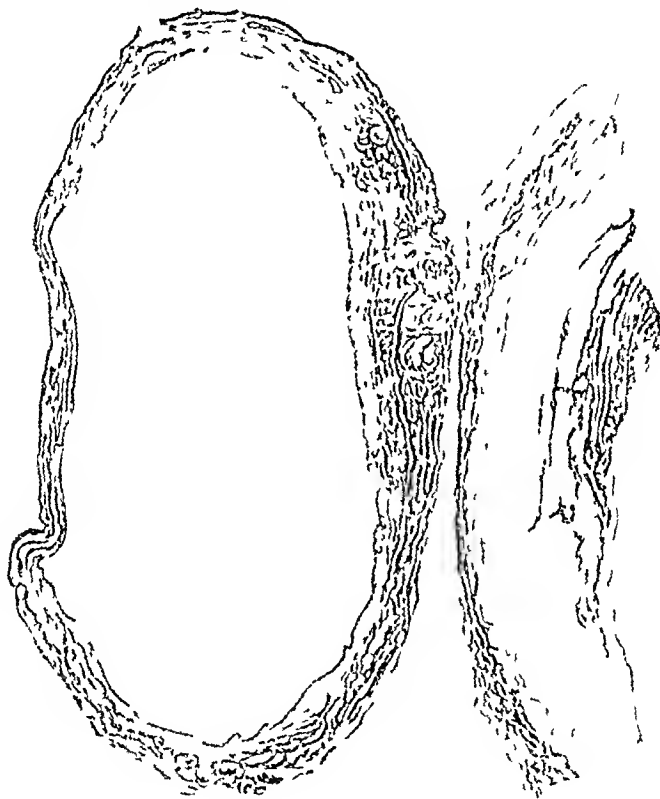


FIG 78—*Case 205* The microscopic appearance of the diverticulum illustrated in *Fig 77*. Only a few muscle fibres are to be seen in the wall of the diverticulum (Van Gieson stain) (5) (M^{rs} S. Steward)

tortuous course. This vessel is best seen on the wall next to the appendix, where the submucosa is at its thickest. The communication between the diverticulum and the lumen of the appendix cannot be seen in this field or in any of the sections taken. It is a very narrow passage, a faint indication of which is seen in the macroscopic view.

The striking feature of the appendix wall adjacent to the diverticulum is the extreme thickness of the muscle coat. It has taken the stain (van Gieson) very poorly, however, and is a much lighter colour than normal. In the higher-power examination there is a lack of definition between the fibres and a scarcity

of nuclei, and there appears to be an inflammatory exudate between the groups of fibres

SUMMARY—The specimen shows an appendix thickened as a result of chronic inflammation. The distal half of the lumen is tortuous, and distended to form a series of pockets, which are retained within the muscle coat. Near the tip is a large diverticulum, whose opening corresponds in situation with a large blood-vessel. The muscularis throughout the appendix, except at one situation where the appendix is kinked, is thickened, probably from hypertrophy, and has undergone a secondary degenerative process. The diverticulum possesses no muscular coat.

Specimen 9 (Case 206)—This specimen (unillustrated) was removed at operation from a woman of 48. It was a short, very stumpy appendix with the walls extremely thickened by fibrous tissue. There were five or six small swellings under the peritoneum in the distal half, with rigid walls. On section the lumen was almost completely obliterated by fibrosis, and the small nodules were solid masses of fibrous tissue. The muscle

FIG 79—Case 206. The microscopic appearance of Specimen 9. The muscular coat of the appendix is extensively broken up by inflammatory fibrous tissue. A, Fibrous tissue, B, Muscle coat.

coat was broken up extensively by fibrous masses extending from the submucosa.

The microscopic appearance in this case is illustrated by a photomicrograph in Fig 79. There is an extensive fibrosis of the submucosa, which has penetrated into the muscle coat, subdividing into small atrophic islands. In the centre the whole thickness of muscle has been invaded by fibrous tissue, and all that remains of this coat is small isolated bundles of fibres buried in great masses of fibrous tissue. There is no clear division between submucosa and muscularis.

SUMMARY—The case illustrates the way in which an inflammatory fibrosis may cause great gaps in the muscle coat through which diverticula may occur.

Specimen 10 (Case 207)—The specimen illustrated in Fig 80 was removed at operation from a man of 55. The appendix is thickened and almost bulbous

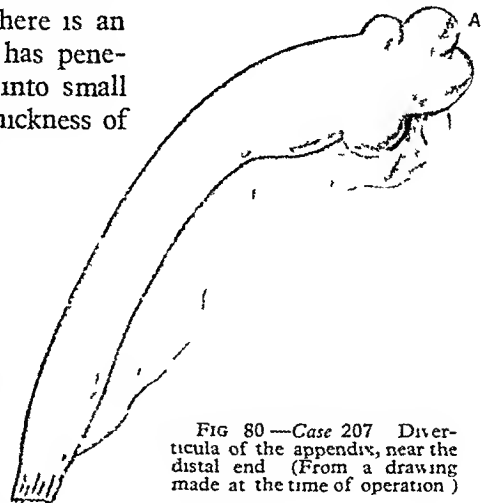


FIG 80—Case 207. Diverticula of the appendix, near the distal end. (From a drawing made at the time of operation.)

towards the distal end. Near the tip are three small diverticula, one of which passes into the meso-appendix, and the other two lie on the free border.

Fig. 81 illustrates a microscopic section passing through the tip of the appendix and the diverticulum marked *A* in *Fig. 80*. The lumen of the mucous



FIG. 81—*Case 207*. Microscopic appearance of a whole section across the tip of the appendix illustrated in *Fig. 80*. The diverticulum opens from the antimesenteric border of the appendix, and contains very little muscle tissue in its wall. The lumen of the appendix is eccentric. (*Mr. S. Steward*.)

membrane is eccentric, and for half its diameter lies in a diverticulum of the normal type.

The muscular wall of the appendix is well developed. The diverticulum passes through a gap in the longitudinal muscle coat, carrying with it the circular muscle coat, which is extremely thin toward the fundus of the diverticulum. The submucous layer is thicker here than at the neck.

Fig 82 illustrates a section from another aspect. It has caught the lower border of the diverticulum. Here the lumen of the appendix is no longer eccentric. The most remarkable feature of this section is the integrity of the circular muscle-fibres in contact with the diverticulum, and the way the longitudinal fibres of the appendix are carried out into the diverticular wall. Note the unusually large gap in both coats at the attachment of the mesentery.

SUMMARY—The mucous membrane has been bodily displaced to one side, and has herniated through a gap in a well developed muscular coat.

PATHOGENESIS

Before arriving at conclusions from the abundance of data offered by these ten specimens, it will be well to attempt a grouping, and to discuss the significance of each group. These are discussed in the following order: (1) The relation of the diverticula to the muscular coat, (2) The inflammatory changes in the appendix wall, (3) The situations of the diverticula in relation to the circumference of the appendix, (4) The changes in the mucous membrane affecting the lumen.

I The Relation of the Diverticula to the Muscular Coat—In all cases the fully formed diverticulum lacks a muscular coat. In the greater number of cases the diverticulum is a true hernia through a gap in the muscle coat. In other cases, such as in *Specimen 5*, where there is distension of the lumen so that pockets are formed, there still

remains a thin layer of muscle, and there can be no doubt that these pockets are the direct result of distension. They cannot be regarded as fully developed diverticula, but rather as sacculations of the lumen. Eventually, if the process were allowed to go on, the muscular coat would completely disappear over the fundus as a result of atrophy, and a fully developed diverticulum, to be seen as a prominence under the peritoneal coat, would result. Thus there are clearly two types of diverticula: (a) Hernial pouches of mucous membrane forced through a gap in the muscle coat, and (b) Distended pockets of the mucous membrane over which the muscularis will eventually atrophy, so that a complete diverticulum, visible from the peritoneal aspect, is formed.

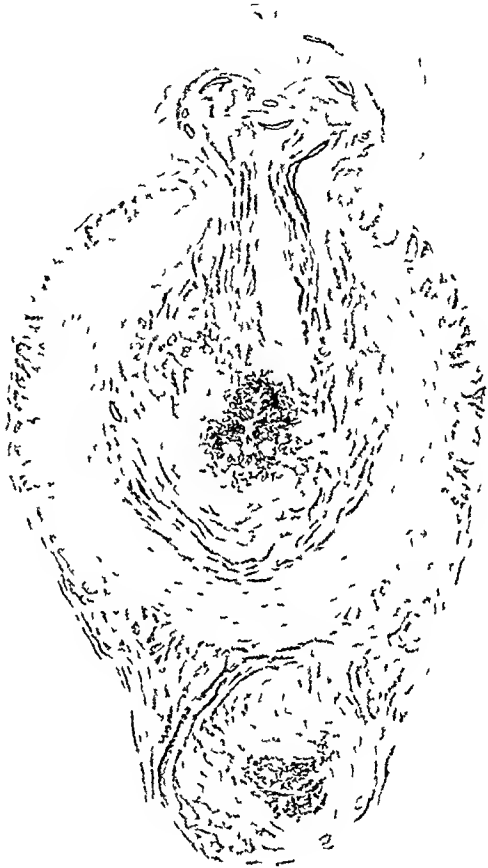


FIG 82—*Case 207*. A further section from *Specimen 10, Fig 80*. The arrangement of the muscular fibres at the base of the diverticulum is well shown. The vascular gap on the mesenteric aspect of the appendix is unusually wide. (*Mr S. Steward*)

2 Inflammatory Changes in the Appendix Wall—All show inflammatory changes. In 7 there is chronic inflammation and in 2 of these acute inflammation has been superadded. In *Specimen 1* the inflammation appears to have been acute from the start, but one cannot be certain of this point. The duration of the history must be given second place in importance in deciding the chronicity of the inflammatory process to the changes found on pathological examination. Thus in *Specimen 7* the history is of only forty-eight hours' duration, but the presence of the concretions in the lumen clearly indicate that pathological change had preceded the onset of subjective symptoms. There is no evidence that the presence of diverticula has been the cause of chronic inflammation. Everything points to the reverse—that the diverticula follow upon inflammation. All of the pouches with the exception of *Specimens 1* and *6* are free from gross inflammatory changes in their walls. In none of them is there any retention of faecal material that might predispose to the onset of inflammatory changes such as occur with diverticulosis of the colon.

In *Specimen 6* two of the diverticula are acutely inflamed, but this does not establish their guilt as the cause of the initial inflammation. It is possible that the perforation in this case took place through a diverticulum, although the area is so destroyed that one cannot be certain on this point. That acute diverticulitis might supervene in diverticulosis of the appendix is, of course, conceivable, and even a likely possibility. Wilkie reports such a case. But this fact is no evidence against the thesis that chronic inflammatory changes in the appendix precede the development of diverticula.

Chronic inflammation in these specimens, at all events in *Specimens 2* to *10*, has aided, possibly been mainly responsible for, the formation of diverticula in one or more of the following ways—

a By causing obstruction to the lumen of the appendix. This has occurred in *Specimens 2, 3, 5, 6, and 8*, the obstruction being somewhere in the proximal half of the lumen. The stenosis in all these cases is marked, but not complete. This is in accordance with what would be expected, as complete obstruction to the appendix at a point near its caecal end would be more likely to result in a mucocoele rather than cause the conditions present in these specimens.

b Chronic inflammatory changes may act by causing weak areas in the muscular coat, through which herniation of the mucous membrane may occur. The sequence of events is probably this. The abundant lymphoid tissue normally present in the appendix is infected, and granulation tissue appears, which invades the submucous coat, and finally the muscularis, leaving a weakened scar. A significant fact is that in many of the microscopic sections made from these specimens the lymphoid tissue is either very scant or absent entirely.

In *Specimen 3* the irregular sites of the small diverticula suggest that the weak areas in the musculature were provided by changes due to chronic inflammation, for they show no tendency to follow the gaps provided by the entrance of the blood-vessels, which will be described subsequently. The microscopic section of *Specimen 9* illustrates how the muscular coat may become disorganized by chronic inflammation.

c Chronic inflammation may be the cause of persistent spasm in the muscular coat, the significance of which is discussed subsequently.

3 The Situations of the Diverticula in relation to the Circumference of the Appendix Wall—The most favoured site for the development of diverticula

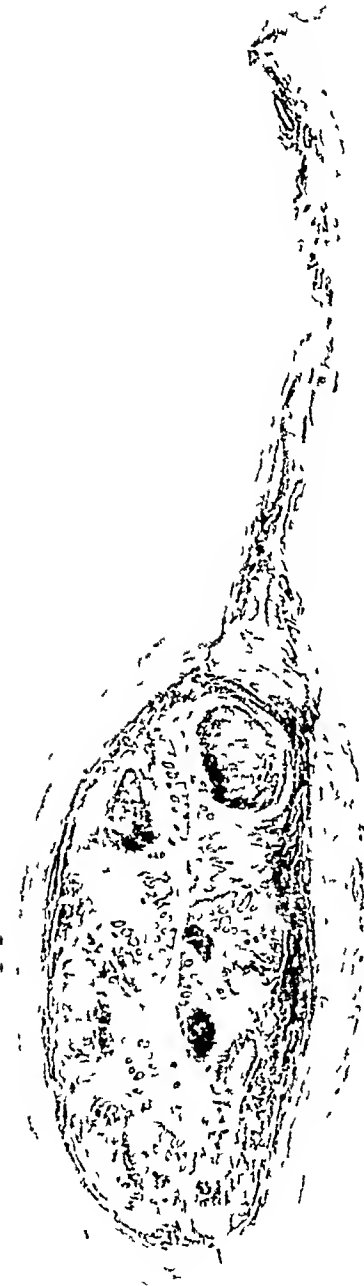


FIG 83—A photomicrograph of a section through the appendix and its mesentery to show the entry of the vessels through the muscular coat (Mr S Sicard)

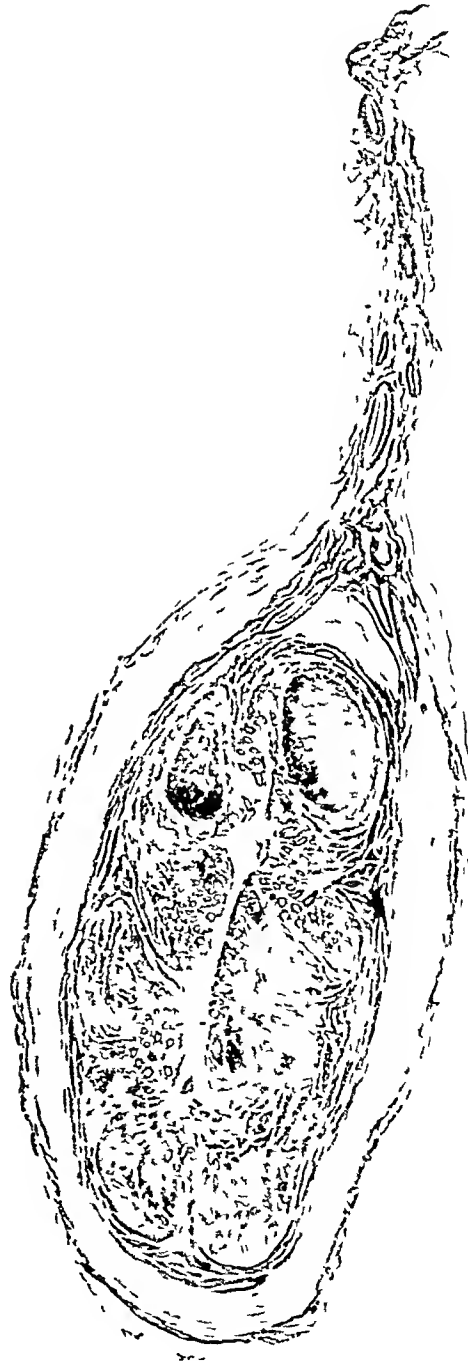


FIG 84—Drawing, to show more clearly the anatomy of the section illustrated in Fig 83 (Miss Barclay-Smith)

is along the concavity of the appendix, along which the vessels of the meso-appendix enter. This is most clearly illustrated in *Specimens 1* and *7*. The next most frequent site is on the opposite (convex) margin.

The gaps caused in the musculature by the entry of the blood-vessels are similar in anatomical features to those of the small intestine. The number of final branches arising from the appendicular arteries depends upon the length of the appendix, varying between 4 and 12.

Immediately before reaching the appendix, each branch divides into two, which pierce the muscle coat, somewhat obliquely, on either side of the mesenteric line. This is shown in the microphotograph of a normal appendix (*Fig 83*) and in the illustration made from the same section (*Fig 84*). *Fig 85* is a diagrammatic representation.

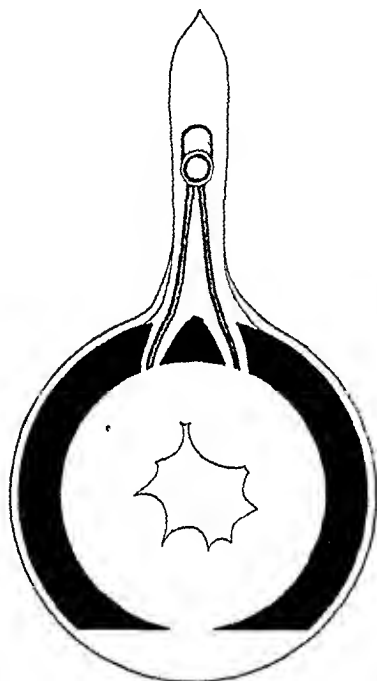


FIG 85 — Diagrammatic representation of the mode of entry of the blood-vessels through the muscle coat

The relation to the entry of vessels of diverticula at the convex margin of the appendix is not so well marked. Before the branches of the appendicular artery enter the concave aspect of the appendix, small branches are given off, which pass on either side around the appendix underneath the serous coat, and enter the muscle layer on the convex margin. According to Stout, these branches do not pass completely through the muscle layer, but terminate in supplying the fibres.

4 Changes in the Mucous Membrane Affecting the Lumen—The most remarkable observation to be made from these specimens is the behaviour, in some of the cases, of the lumen of the appendix.

In *Specimens 4* to *8* the mucosa has been thrown into folds. In between these folds are deep crypts, which in some cases—for example, in *Specimen 4*—have been distended to form wide-mouthed pouches. What is the explanation of this pleating of the mucosa? There clearly has been a relative increase in length of the mucosa and submucosa coat as compared with the length of the muscular and serous coats. Is this the result of actual lengthening of the mucosa or of shortening of the subjacent muscularis? There is no reason to think that the mucosa should increase in length, and the appearances support the view that the folds in the mucosa are due to shortening of the muscular coat. Under normal conditions the attachment between the submucous coat and the muscularis is by loose bands of areolar and connective tissue, so that there can be a considerable excursion of one upon the other.

The muscle coats in all these specimens (*4* to *8*) is very much thicker than in the normal. In ten normal appendices the average thickness of the muscle coat determined by measurement with fine-pointed dividers was approximately 0.75 mm. In these five specimens, the muscle layer is much thicker, varying between 1.5 and 2.5 mm. This increase in thickness may be due to at least one of three

causes (a) Contraction of the muscle and fixation in contraction, (b) Hypertrophy, and (c) Inflammatory œdema

The last-named certainly occurs, but cannot be responsible in all the cases, and one is left with (a) and (b) If this thickness is due to hypertrophy, one would expect to find a partial obstruction present in every case, and the alternative explanation must therefore be accepted The pouching of the mucous membrane, which may lead to diverticula, is due to shortening of the muscle coat as a result of persistent contraction To thickening of the coat from this cause must be added a secondary hypertrophy in most of the cases, in which obstruction has been caused by the formation of these folds in the mucous membrane Migration of the whole lumen to one or other side is associated with this pouching in *Specimens 4, 5, 7, and 8* It is best shown in 5 and 7 Here both layers of the mucous membrane are carried right through the muscle coat into a diverticulum In both cases this has caused a partial obstruction, with subsequent distension of the lumen distal to this point This phenomenon of migration of the lumen is difficult to explain It clearly cannot result from high pressure inside the lumen, and no outside traction could drag both surfaces through the muscular wall of the appendix One must therefore turn to aberrations in the normal action of the muscularis as the cause The probable way in which this acts is demonstrated by diagrams in *Fig 86*

There is first tonic spasm of the longitudinal fibres, so that the mucous membrane is thrown into folds Such spasm of the longitudinal fibres will increase the vascular gaps Spasm of the circular muscle now follows, and the potential cavity of the appendix is obliterated, the surfaces of the lining mucous membrane being pressed together, and lying in contact When such spasm occurs, the loose folds of mucous membrane, lying in contact, will seek an outlet, and both surfaces may pass into a gap occasioned by the passage of a blood-vessel on the concave aspect Once this has been achieved, there will be a tendency for it to become permanent, and by causing a marked obstruction to the outflow of contents from the lumen distal to it, to cause distension of the lumen with pouching of the folds of mucous membrane Such distension may cause herniations through vascular gaps, or cause thinning of the muscularis over the pouches from atrophy so that fully developed diverticula may be formed from them

Specimen 7 illustrates perfectly how crypts of mucous membrane caused by contraction of the longitudinal muscle may pass through the gaps in the musculature on the concave aspect

Such spastic conditions of the musculature may result from lack of neuromuscular co-ordination, such as seems to form diverticula in the small and

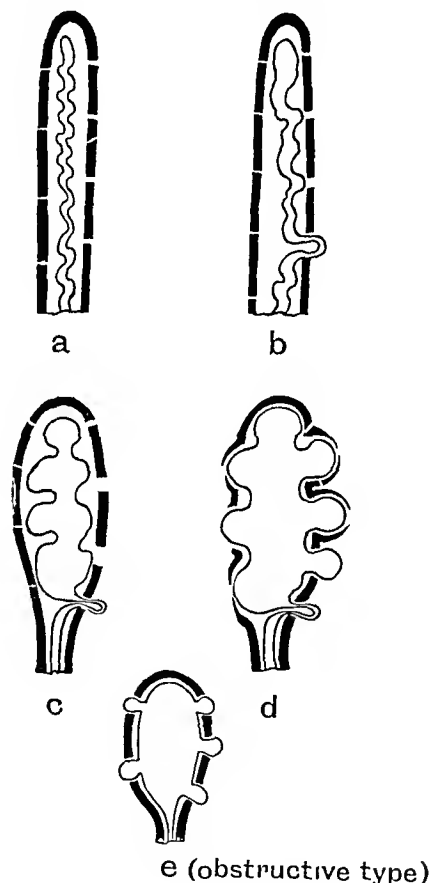


FIG 86 —The mechanism of formation of diverticula of the appendix

large bowels, or may have a more local origin in chronic inflammation of the appendix wall

Commentary—From this seeming tangle of facts, the explanation of the pathogenesis of diverticula of the appendix begins to unfold itself. In not all the cases are the mechanics of production the same, and we can distinguish at least two types —

1 A hernial protrusion of the mucous membrane through a gap in the musculature, occasioned in most cases by the passage of a blood-vessel, and in some as a result of local degeneration of the muscle

2 Distension of sacculations in the mucosa, with subsequent thinning from atrophy of the muscularis covering them

The first type may arise purely as a result of partial obstruction to the lumen. *Specimen 2* is an excellent example of hernial diverticulum as the result of obstruction. The second type—sacculation of the mucous membrane—is a more intricate process, and is indirectly due to spasm of the muscularis, with tortuosity and migration of the lumen (*Specimens 4 to 8*, and ²3). Subsequently, in these cases, obstructive effects are produced, so that the mucous membrane may be forced into gaps in the musculature. Thus diverticula of the appendix may be due to one of two exciting causes: (1) Passive distension, (2) Irregular muscular action. The predisposing causes are: (1) The presence of gaps in the muscular coat through which the vessels enter, and (2) Weakening of the muscular coat as the result of chronic inflammation.

In both the cases diagnosed radiologically the whole colon is also affected. That there is no obstruction to the appendix acting as a cause of pressure within the lumen is proved by the entry of barium. The formation of the diverticula in both the colon and the appendix is undoubtedly due to the same factors, and the knowledge gained of the myogenic origin of diverticula of the appendix is a further link in the chain of evidence that the author has brought forward to substantiate the thesis that hernial diverticula of the alimentary tract owe their origin to irregular contraction of the muscularis (*see BIBLIOGRAPHY*, p. 107).

THE LITERATURE

Pack and Scharnagel describe a case with thirty-six diverticula, and concluded that these resulted from an exudative inflammation which caused disorganization of the muscle coat. Wilkie describes two cases associated with obstructive carcinoma of the appendix. In one of these perforation occurred through a diverticulum. Chase thinks the gap in the musculature—which he calls “hiatus muscularis”—may occur from the passage of smaller vessels and lymphatics or be an interval occupied solely by areolar tissue or fat. He recognizes two types: simple herniation of the mucous membrane through a gap, and diverticula due to chronic inflammation. The article of MacCarty and McGrath has already been referred to. Stout has shown by experiment upon the living dog that if a gap be made in the muscular wall of the appendix, the mucosa and submucosa are protruded. In one such experiment he excised a portion of the serous coat and muscular coat measuring 6 mm. by 8 mm. The appendix contracted both in diameter and length and became very hard, and the mucous membrane was forced through the gap to form a prominence 4 mm. in height. When the muscle coat

relaxed again the mucous membrane pouch became smaller, but did not disappear completely. Moderate hydrostatic distension made the diverticulum stand out more clearly as a smooth rounded hemisphere. When the hydrostatic pressure was increased further, the coats of the appendix slowly thinned from stretching, and the gap in the muscularis became wider. The diverticulum grew steadily less until it disappeared entirely. Such an effect is produced by the internal pressure overcoming the force of muscular contraction.

From this and similar experiments Stout concluded that diverticula may be produced both by muscular action and by increased intraluminal pressure. These views correspond with the deductions made from the author's series of pathological specimens.

DIAGNOSIS AND TREATMENT

There are no symptoms directly referable to the presence of diverticula, so that it is not possible to diagnose the condition prior to operation. Only

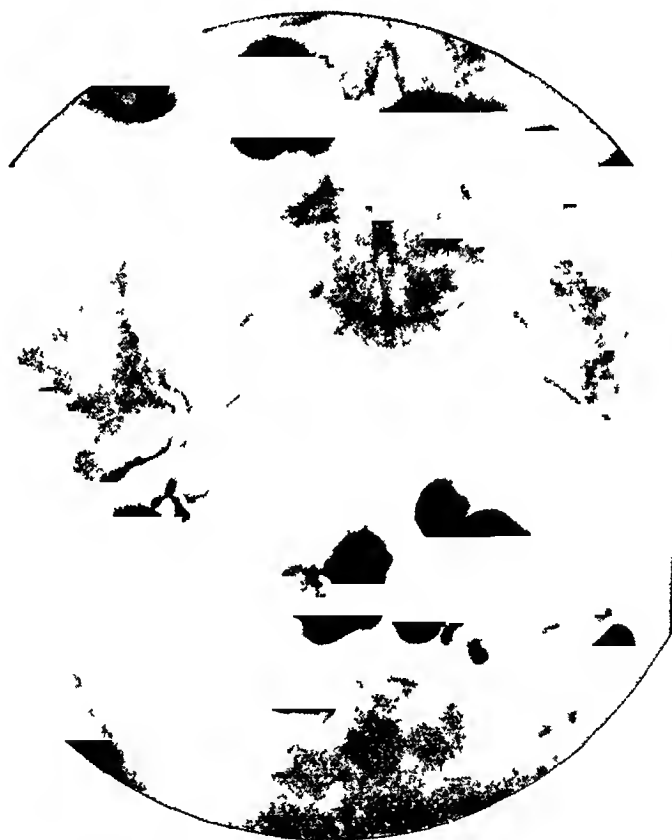


FIG 87—Case 209 Diverticula of the appendix, demonstrated radiologically. Diverticula were also present in the colon (Dr Graham-Hodgson)

very rarely, as in the two cases described, do diverticula show up on X-ray examination. The appearance of one of the cases is illustrated by a skiagram

of a barium meal taken thirty-six hours after ingestion (*Fig 87*) The treatment is appendicectomy

UNUSUAL FORMS AND PSEUDO-DIVERTICULA

Fig 88 illustrates an appendix in which there is a narrowing of the lumen $\frac{1}{3}$ in from the tip The appendix is otherwise normal The distal pocket is somewhat distended and its wall a little thickened The case cannot be regarded as a diverticulum in the true sense of the word There is no 'turning aside' (*Case 208*)

The illustration in *Fig 89* was copied directly from an article by Herb The excuse offered for making such a copy is that words could not readily convey the



FIG 88 — A pseudo diverticulum of the appendix The distal part of the lumen is shut off by a transverse band (*Miss Barclay-Smith*)

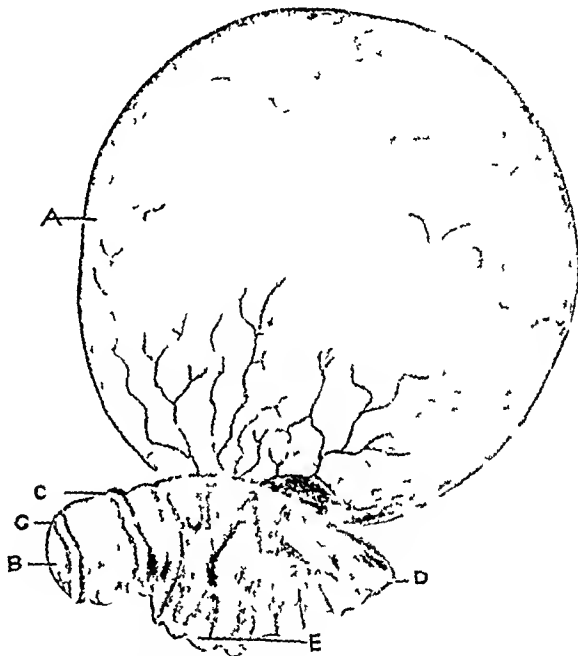


FIG 89 — An enormous diverticulum of the appendix, copied from an illustration of the case described by Herb A, Diverticulum, B, Proximal end of appendix, showing closure, C, Retracted muscular coats, D, Distal end of appendix, E Meso-appendix The lighter areas on the diverticulum are calcified plaques

remarkable condition which is pictured The enormous swelling arising from the convex surface of the appendix is 9 in in circumference, and communicates with the lumen of the latter by a circular opening 1 in wide The lumen of the appendix is obstructed at the proximal end The sac was filled with jelly-like substance Its wall consists of mucosa and fibrous tissue only, there being no muscle and no mucous membrane The muscularis of the appendix ends abruptly at the margin of the sac This, though unquestionably a type of diverticulum, belongs to the little understood condition described as mucocoele of the appendix

My thanks are due to the Council of the Royal College of Surgeons for permission to publish this section of the Jacksonian Prize Essay for 1932

I wish also to thank my colleagues on the staff of King's College Hospital for permission to use cases operated upon by them, and Miss Mary Barclay-Smith and Mr S Steward, who are responsible for the majority of the illustrations

BIBLIOGRAPHY

- CHASE, W H, *Canad Med Assoc Jour* 1927 **xvii**, 416
 EDWARDS, H C *Lancet*, 1934, **i** 169, 221
 HERB, I C, *Trans Chicago Pathol Soc*, 1907, **vii** 94
 MACCARTY, H C, and McGRATH, B F *Surg, Gynecol and Obst* 1911, **xii** 211
 MOSCHOWITZ, E, *Ann of Surg* 1916, **lxiii**, 697
 PACK, G T and SCHARNAGEL I, *Amer Jour Surg* 1928, **v** 369
 STOUT, A P A, *Arch of Surg*, 1923 **vi** 793
 WILKIE D P D, *Brit Jour Surg*, 1921, **viii**, 392

ACUTE ENTERIC INTUSSUSCEPTION IN AN ADULT CAUSED BY A LIPOMA

WITH A SURVEY OF THE LITERATURE

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THE case described below warrants publication in that it presents the following features (1) It is a case of intussusception in an adult, (2) It is of the pure enteric variety, small bowel only being involved, (3) The cause is a benign tumour, a submucous lipoma, (4) The specimen (*see Fig 90*) illustrates the modern view of the physics of intussusception, (5) After resection and entero-anastomosis the patient made a complete recovery

CASE REPORT

T B, aged 52 years, a coal heaver, was admitted to Boundary Park Municipal Hospital, Oldham, on Sunday, March 8, 1931, suffering from intestinal obstruction

HISTORY—The patient's previous medical history was unimportant except for an operation for anal fissure in 1928. He gave a history of having been taken ill on the previous Thursday with severe cramp-like pains in the abdomen and he went to bed on that day. He vomited frequently and the pains became worse. He had passed neither *faeces* nor *flatus* since the onset of his illness.

ON EXAMINATION—He looked ill and 'toxic', and at midnight his temperature was 97°, the pulse 92, and the respiratory rate 20. The abdomen was distended, tender all over, and liver dullness had disappeared. There was dullness in both flanks. Nothing could be felt per rectum and the examining finger was not blood-stained. Immediate operation was decided upon.

OPERATION—Under a general anæsthetic a mass could be felt to the right of and below the umbilicus. The abdomen was opened through a right paramedian incision and a large quantity of blood-stained fluid escaped. The mass proved to be an intussusception composed entirely of small intestine. The intussusciptens was gangrenous and covered with plastic lymph. A very gentle attempt at reduction caused the outer coat to tear badly. Resection was decided upon, and the whole mass was rapidly removed and a lateral anastomosis made between two convenient loops of small intestine. The abdomen was then swabbed out. As the patient showed signs of collapse no attempt was made to close the abdomen in layers, six silkworm gut through-and-through sutures only being used. The usual measures were employed in the immediate after-treatment.

SUBSEQUENT PROGRESS—The patient was very ill for some days, severe incontinence being the most striking feature. Anti-gas-gangrene serum was given daily for a week. The incontinence gradually subsided and the wound broke down and discharged in several places, but at the end of a week the patient began to improve, the wound commenced to heal, and the bowels became normal in action.

He was discharged from the hospital soundly healed, feeling well and able to walk, six weeks after admission. He is now engaged in his former occupation as a coal heaver and feels perfectly well.

PATHOLOGICAL REPORT (*Fig 90*)—The tumour projecting into the lumen of the small intestine has a nodular surface. It is attached by a pedicle of fibrous tissue and is enclosed

in a fibrous capsule. On section it is pale yellow in colour and firm in consistence. It is roughly spherical in form and about 2 cm in diameter. The mesentery is inflamed and there are definite signs of peritonitis. Histologically the tumour is a pure lipoma, staining uniformly with Sudan III.

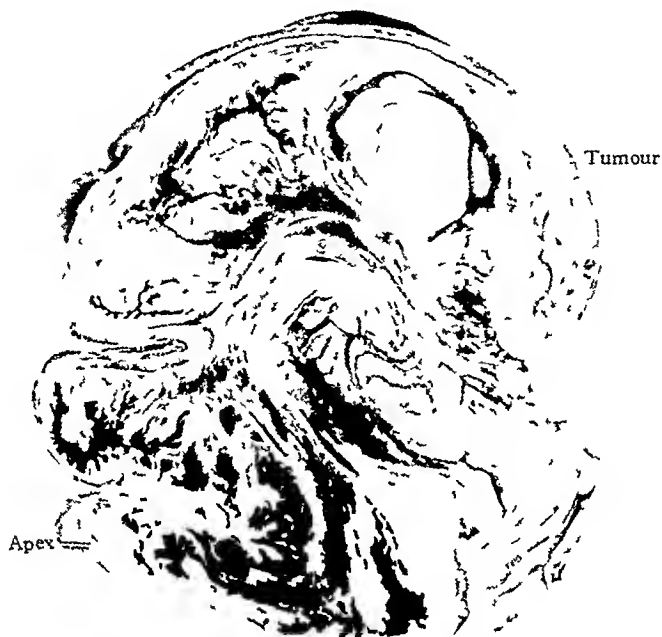


FIG 90—A portion of the intussusciens is not distinctly shown in the photograph. As indicated in the report of the case, the receiving layer was gangrenous and tore on an attempt at reduction, and whilst in the preserving fluid the torn and gangrenous portion curled away behind the plane of section.

INTUSSUSCEPTION IN ADULTS

To those associated with large teaching and children's hospitals intussusception is fairly common, yet taking the ordinary incidence of the condition in relation to the population it is rare. The writer is acquainted with a very able general practitioner who never even saw a suspicious case in twenty-five years' practice.

During the last decade there have been 27 cases (one of which recurred) operated upon at the Oldham Royal Infirmary, 10 cases at the Boundary Park Municipal Hospital, and 1 at a local nursing home—a total of 38 cases in ten years amongst a population of about a quarter of a million served by these institutions. Of these cases, only one, the writer's, was an adult.

Choyce⁴ states that not more than 12 per cent of all cases of intussusception occur in patients over 10 years old. Glover,¹¹ in reporting a case of enteric intussusception in a man of 47 with no indication as to cause, comments on the rarity.

The majority of cases of invagination in adults appear to be due to a growth of a benign character. Muller²⁶ describes one as due to a mucous papilloma. Davies⁶ and Dewis⁸ describe cases due to fibroma, and Somerville-Large³³ one due to a polypus. As a malignant cause Morrison²⁹ has recorded a case due to a lymphosarcoma. Lenner¹⁸ and McIver²⁰ have each described cases due to Meckel's diverticula. Ruppaner³² quotes a very unusual one following gastro-enterostomy.

Mesnager²² notes a case apparently due to a fall, and refers to a similar case recorded by Kopp Moor²⁴ describes two cases occurring in Mohammedans during a fast, and points out that excessive hunger associated with the anticipation of a meal can excite abnormal peristalsis. A case due to a tuberculous intestine in a boy of 16 has been recorded by Sussig,³⁷ whilst Thorleifson³⁸ has described a case of intussusception of the jejunum where no cause could be found at autopsy.

LIPOMATA OF THE GASTRO-INTESTINAL TRACT

There is much overlapping in the literature connected with this subject. Previously reported cases have been overlooked by some writers, so an attempt is made below to collect and correlate reports from all sources. Using as a basis the paper by Comfort,⁵ the writer was able to trace the following —

Comfort, own and collected cases	96
Mirolli, ²³ cases not included above	103
Stettin, ³⁶ cases not included above	14
White and Judd, ¹⁰ cases not included above	13
Writer's case and other cases not included above	16
Total	242

The cases found by the writer are reported by Bailey,¹ the BRITISH JOURNAL OF SURGERY SUPPLEMENT,² Brandes,³ Form,¹⁰ Farr,⁹ Horsley,¹² McCloskey,¹⁰ Matry,²¹ Neumann,²⁷ Oughterson and Cheever,²⁸ Polya³⁰ (two cases), Rouse and Mekie,³¹ and Stewart and Illick³⁶ (two cases).

The anatomical distribution of the tumours was as follows —

Stomach	27	Sigmoid colon	21
Duodenum	18	Rectum	11
Small intestine	64	Mesentery	1
Ileocaecal region	10	Incomplete accounts	11
Cæcum	20		
Colon	59	Total	242

Intussusception is reported to have occurred in 80 cases in the following sites —

Small intestine	34	Sigmoid colon	10
Ileocaecal region	5	Uncertain	5
Cæcum	6		
Colon	20	Total	80

It will be seen therefore that not only is lipoma of the gastro-intestinal tract an uncommon condition, but also that enteric intussusception due to this benign new growth is very rare.

MECHANICS OF INTUSSUSCEPTION

Our surgical forefathers cannot be blamed for the unwieldy name given to this condition. A 'reception within' warrants some preparation and something to receive. The writer has been unable to trace any case where a foreign body has caused invagination, a foreign body is either passed per rectum or it causes perforation or obstruction of the bowel. It would appear, then, that it is only

when that which is to be received is part of the bowel itself or attached to it that actual invagination occurs, and this presumption is borne out not only by the physiological but also by the pathological facts

Starling³⁴ describes normal peristalsis as a slowly moving wave, generally at about 2 cm a minute, with contraction of the gut above the food mass and relaxation below it. Illingworth and Dick¹⁶ believe that there is considerable evidence to show that the underlying cause of intussusception is some derangement of this peristaltic mechanism excited in children by errors in diet. Such errors are notorious between the fourth and seventh month, but it is also pointed out that the inhibitory nervous apparatus is developed last and that its action is apt to lag behind that of the motor apparatus. In infancy, therefore, the inhibiting activities of the parasympathetic nerves are functionally weak and the tonicity of the plain muscle outweighs, for a time at least, its capacity for relaxation.

Comparing the intussusception of the infant with that of the adult, Hubner¹³ points out that intestinal disorders in the infant, such as colitis, seem to have no great influence, as nearly all cases occur in infants in good health. A *terram maladi* is not necessary as a background. He quotes Withner, who noted that a tumour was found in only 2 cases out of 200 infants suffering from intussusception. Hubner believes that if a peristaltic wave starts at a given point it is accompanied always by an inhibition in the segment below this point. This lower segment relaxes under the influence of a stimulus brought upon the given point, whilst above the point there is a reinforcement of the contraction. With a normal intestinal content only a minimum muscular effort is required, contraction following upon dilatation, and so on. If, however, from some cause the ring of contraction becomes fixed and immobile, one can conceive that this fixed and immobile part of the intestine can penetrate into the dilated portion below it and so form the beginning of the invagination. Nothnagel is quoted as having actually produced invagination by electric stimulation of the bowel itself. Therefore "the *agent provocateur* of the exaggerated peristalsis on the one hand is, on the other, the fixing agent."

Dewis⁸ noted that while the tumour is usually situated near the apex of the invaginated mass, it is occasionally attached to other portions of the mass. It is pointed out by Wardill³⁹ that when a polyp of bowel is associated with an intussusception there is a definite relationship of cause and effect. He has found that specimens show that tumours may occupy a position some considerable distance proximal to the apex although still included in the intussusception. (This important point is very well illustrated in the writer's own case.) Mere traction on the polypus by intestinal movements does not account for the commencement of the invagination. The tumour lies within the intestinal lumen and so acts as a foreign body which produces spasmodic contraction of the gut around it with inhibition of the gut immediately distal to it. "The conditions are now favourable for that act of peristaltic gymnastics whereby the contracted part is induced to slip into the dilated portion." Here is the whole physiological and pathological explanation in a nutshell.

Discussing the problem at a later date, Iason and Filberbaum¹⁴ go into the causes of intussusception in adults. Accepting Wardill's theory, they state that other causes may be (1) Passive, due to the mere weight and pull of the tumour, (2) Active, owing to the violent peristalsis due to the normal reaction to a foreign

body in the lumen, (3) Perverted muscle action due to the presence of a tumour, and (4) Paralysis of the bowel itself

Ibos and Legrand-Desmons,¹⁵ in discussing a case of retrograde intussusception of the small intestine, are of the opinion that their case proves that a pathological antiperistalsis can occur in the intestine

DIAGNOSIS

The diagnosis of a benign new growth in the alimentary canal must always be a matter of difficulty, and usually its presence is not suspected until a laparotomy is performed for some condition of which it may or may not be the cause. Pollosson and Rougemont²⁹ have described a syndrome which is worth quoting as it offers a fairly definite clinical picture. The patient has several attacks of abdominal pain of such severity that he remembers each attack. He suffers from borborygmi and the colicky pains come on at regular intervals. There is complete abdominal relaxation with no distension and no rigidity. The pulse and temperature remain normal. If a tumour is felt at this stage, appendicitis or a similar inflammatory condition can be ruled out, as such conditions cannot form a swelling in a few hours. Examination of the urine may help to exclude renal colic. These writers emphasize the importance of abdominal palpation under an anæsthetic (This proved of value in the writer's own case). With the onset of hæmorrhage, obstruction, or intussusception the symptoms due to these conditions will appear.

In this connection Wardill³⁰ stresses the point that when operating for intussusception, particularly in subjects over the age of 2 years, a careful examination should be made of the *proximal healthy bowel* (the italics are mine) for possible new growth. Hubner¹³ makes a similar observation, but states that in all cases where an intestinal tumour was noted, an acute intussusception was the first sign of its existence. Iason and Filberbaum¹¹ point out that the appearance of blood per rectum is less frequent in the intussusception of adults than in that of children, and if present points to torsion or ulceration of the tumour. (It will be noted that the examining finger showed no blood in the writer's case.) The probable explanation of this is that when the invagination is high up coagulation takes place.

Before passing from the consideration of the diagnosis of benign intestinal tumours it is well to remember that these tumours are often discovered during the so-called 'cancer age'.

TREATMENT

In the treatment of intussusception Leclerc¹⁷ recommends attempts at disinvagination in the first place, and, if this fails, resection either of the invaginated cylinder of gut or of the whole of the affected bowel with entero-anastomosis. In desperate cases the formation of an artificial anus or 'exteriorization' of the affected gut with 'fistulization' is urged. Delore and de Girardier⁷ believe that resection of the 'black pudding' through the receiving layer is often enough. Finally, Oughterson and Cheever²⁸ strongly advocate that every tumour of the intestinal tract, especially if it projects into the intestinal lumen, should be removed no matter how benign the character, unless there is some definite contra-indication, since it always carries with it the threat of intussusception.

SUMMARY

- 1 A case is described of acute enteric intussusception in a man aged 52 years
- 2 The cause of the intussusception was a submucous lipoma
- 3 A search of the literature has shown that there are on record 242 cases of lipomata of the gastro-intestinal tract, 64 of these occurred in the small intestine
- 4 Eighty cases of intussusception have been described as being due to lipomata, 34 being enteric in variety
- 5 The case under consideration illustrates the modern theories of the mechanics of intussusception. These theories indicate that the tumour is not necessarily at the apex of the invagination
- 6 Such a tumour is usually not discovered until it causes symptoms or until a laparotomy is performed for some condition of which it may or may not be the cause
- 7 The treatment of intussusception due to a tumour is along the usual surgical lines, but search for a tumour especially in the proximal gut should be made in all cases of intussusception occurring after infancy, and, conversely, all benign tumours of the alimentary canal should be removed as being potential causes of intussusception

I am grateful to Dr R P Parker and Dr R A Jackson for permission to publish this case, and to Dr Raymond Whitehead for the great care he has taken in preparing, mounting, and photographing the specimen

REFERENCES

- ¹ BAILEY, "Acute Intussusception in Adults", *Birmingham Med Rev*, 1928, III, 287
- ² *Brit Jour Surg Supplement*, 1933, No 32, 269
- ³ BRANDES, "Ueber Dunndarmdivertikel. Nebst einem Beitrag zur Beobachtung eines hochsitzenden Dunndarmdivertikels bei einem Darmlipom", *Deut Zeits f Chir*, 1929, CCXV, 390
- ⁴ CHOYCE, *A System of Surgery*, 1932
- ⁵ COMFORT, "Submucous Lipomata of the Gastro-intestinal Tract. Report of Twenty-eight Cases", *Surg Gynecol and Obst*, 1931, LI, 101
- ⁶ DAVIES, "Ileo-ileal Intussusception due to Fibroma", *Brit Med Jour*, 1929, I, 446
- ⁷ DELORE and DE GIRARDIER, "Au Sujet du Traitement de l'Invagination intestinale chez l'Adulte, de la Resection du Boudin a travers la Gaine", *Jour de Chir*, 1930, XXXV, 641
- ⁸ DEWIS, "Small Fibroma of the Ileum resulting in Obstruction of the Bowel, with a Consideration of Various Forms of Benign Intestinal Tumours", *Boston Med and Surg Jour*, 1906, CLV, 427
- ⁹ FARR, "Lipoma of Colon", *Surg Clin N Amer*, 1930, V, 477
- ¹⁰ FORNI, "Invaginazione cronica remittente del Digiuno da Lipoma intraparietale. Encerectomia. Guarigione", *Ann ital di Chir*, 1930, IX, 556
- ¹¹ GLOVER, "Enteric Intussusception", *Brit Med Jour*, 1931, I, 58
- ¹² HORSLEY, "Intussusception due to Intestinal Lipoma in Adult followed by Gangrene in Abdominal Wall. Plastic Operation for Repair of Abdominal Wall", *Arch of Surg*, 1929, LVIII, 882
- ¹³ HUBNER, "Invagination intestinale aigue de l'Adulte et de l'Adolescent", *Gaz des Hôp*, 1929, CII, 257, 293
- ¹⁴ IASON and FILBERBAUM, "Intussusception in an Adult associated with Adenoma of Ileum", *Ann of Surg*, 1931, XCII, 1191
- ¹⁵ IBOS and LEGRAND-DESMONS, "Invagination jejunaie aigue chez un Adulte", *Bull et Mem Soc nat de Chir*, 1929, LV, 1277

- ¹⁶ ILLINGWORTH and DICK, *Text-book of Surgical Pathology*, 1932
- ¹⁷ LECLERC, "Traitement de l'Invagination intestinale aigue chez l'Adulte", *Presse med*, 1929, xxxvii, 324
- ¹⁸ LENNER, "Ueber akute Invagination des Dunndarms im Kindesalter, ausgelost durch ein umgestulptes Meckelsches Divertikel", *Beitr z klin Chir*, 1930, cxlix, 631
- ¹⁹ MCCLOSKEY, "Lipoma of Small Intestine (Ileum), with Superimposed Ulceration", *Pennsylvania Med Jour*, 1931, xxxiv, 713
- ²⁰ MCIVER, "Intussusception of Small Intestine with Special Reference to Meckel's Diverticulum as Causative Factor", *New Eng Jour Med*, 1928, cxix, 453
- ²¹ MATRY, "Lipoma du Cæcum Invagination Colectomie droite Guérison", *Bull et Mem Soc nat de Chir*, 1928, liv, 1375
- ²² MESNAGER, "Invagination aigue, retrograde, de Grêle chez l'Adulte", *Bull et Mem Soc Chir de Paris*, 1929, xvi, 788
- ²³ MIROLI, "Contributo allo Studio dei Lipomi sottomucosi e sotto sierosi dell' Intestine, Illustrazione di un Caso con Eliminazione spontanea del Tumora", *Arch ital di Chir*, 1929, xxiv, 553
- ²⁴ MOORO, "Acute Intussusception in Adults during a Fast", *Brit Med Jour*, 1924, i, 319
- ²⁵ MORRISON, "Case of Double Intussusception from Tumour of Terminal Ileum", *Surg Clin N Amer*, 1928, viii, 1473
- ²⁶ MULLER, "Ueber einen Fall von seithcher duodenal Invagination durch Duodenalpolypen mit gleichzeitigen Iso- und Antiperistaltischen Invaginationen des Dunndarms", *Arch f klin Chir*, 1929, cli, 493
- ²⁷ NEUMANN, "Lipome des Magens", *Zentralb f Chir*, 1930, lvii, 1154
- ²⁸ OUGHTERSON and CHEEVER, "Recurring Intussusception caused by Intestinal Neoplasms, requiring Multiple Operations for its Relief", *Surg Gynecol and Obst*, 1929, xlviii, 682
- ²⁹ POLLOSSON and DE ROUGEMONT, "A propos de l'Invagination intestinale chez l'Adulte", *Presse med*, 1928, xxxvi, 278
- ³⁰ POLYA, "Zur Kenntnis der Darmlipome", *Zentralb f Chir*, 1929, lvi, 2518
- ³¹ ROUSE and MEKIE, "Case of Intestinal Lipoma", *Edin Med Jour*, 1930, xxxvii, 50
- ³² RUPPANNER, "Invaginationsileus nach Gastroenterostomie", *Schweiz med Woch*, 1929, lix, 1237
- ³³ SOMERVILLE-LARGE, "Double Intussusception of Jejunum associated with Polypus", *Brit Jour Surg*, 1929, xvi, 340
- ³⁴ STARLING, *Principles of Human Physiology*, 1930
- ³⁵ STETTIN, "The Submucous Lipoma of the Gastro-intestinal Tract", *Surg Gynecol and Obst*, 1909, ix, 156
- ³⁶ STEWART and ILICK, "Lipoma of Colon, Report of Two Cases", *Amer Jour Roentgenol*, 1930, xxiii, 308
- ³⁷ SUSSIG, "Un Caso di Tuberculosis intestinale come Causa di Invaginazione enterica ascendente", *Gaz deg Osped*, 1928, xli, 1079
- ³⁸ THORLEIFSON, "Case of Intussusception of Jejunum", *Canad Med Assoc Jour*, 1929, xx, 395
- ³⁹ WARDILL, "Polypi of the Bowel causing Intussusception", *Brit Jour Surg*, 1925, xiii, 158
- ⁴⁰ WHITE and JUDD, "Lipoma of Stomach Report of Case", *Amer Jour Surg*, 1929, vi, 662

CYSTS OF THE EXTERNAL CARTILAGE OF THE KNEE WITH EROSION OF THE HEAD OF THE TIBIA

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THE two following cases appear worthy of record —

Case 1 —Mr R, aged 49 Referred by Dr Roberts, of Farnham, in February, 1930, for trouble in the left knee

HISTORY —Symptoms dated from a football injury in 1903, after which the knee swelled The knee was weak for years and liable to go inwards and to give



FIG 91—*Case 1* Cyst of the external cartilage of the knee eroding the head of the tibia of a man aged 49

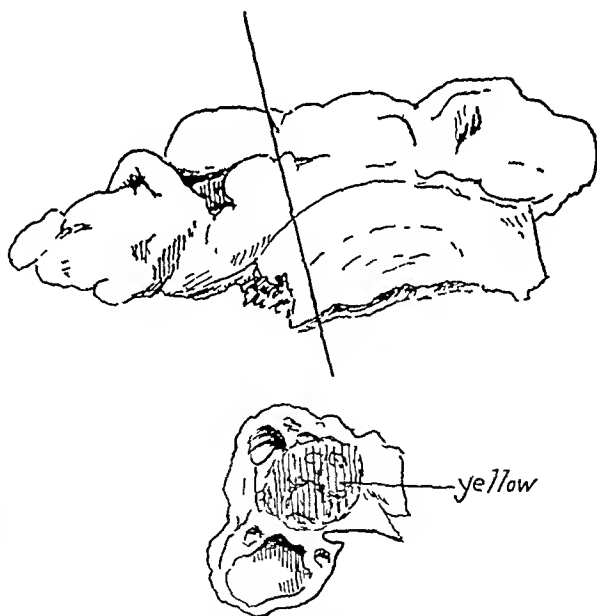


FIG 92—*Case 1* Cyst of the external cartilage of the knee (Natural size)

sharp twinges of pain on the inner side, never locked It eventually got well and remained well for many years The patient was able to play tennis but dared not jump In 1925 he began to have pain on the outer side of the knee, the pain extending upwards and downwards on the outer side of the leg There was a tender spot, and later a swelling appeared The swelling is always present, but varies in size Every six or eight weeks it gets worse, more swollen, and more tender Increase of symptoms definitely due to excessive walking When bad,

knee aches at night It is stiff after sitting for long The patient's doctor burst the swelling by firm pressure last year, and this relieved the patient at once

ON EXAMINATION—There was a typical elastic swelling, slightly tender, beneath and just in front of the external lateral ligament Definite lipping of the bones X-rays showed definite osteo-arthritic changes with a punched-out notch immediately below the outer margin of the tibia, between this and the head of the fibula (*Fig 91*)

OPERATION (March 28, 1930—H A T F)—An unusually large multilocular cyst of the external cartilage was removed together with rather more than the anterior half of the external semilunar cartilage There was a deep groove in the tibia from which the cystic mass was shelled, leaving bare bone, at the site shown in the X-ray The groove was quite smooth and obviously due to pressure

PATHOLOGICAL REPORT—Examination of mass removed (*Fig 92*) showed the mass of cysts extending right to the anterior extremity of the cartilage as well as beyond the line of the external lateral ligament The centre of the tumour was bright yellow, with a fibrous network in it The rest showed the usual fibrocystic appearance The semilunar cartilage was split horizontally throughout its length Microscopical section confirmed the diagnosis

SUBSEQUENT HISTORY—A recent report states that there has been no recurrence of the swelling or symptoms



FIG 93—*Case 2* Cyst of the external cartilage of the knee eroding the head of the tibia of a man aged 36 There is also an old stellate fracture of the patella

Case 2—Mr W M, aged 36, had been troubled by his right knee for fifteen years and a swelling had been noticed for ten years He attributed his symptoms to a fall which was followed six months later by a kick The swelling was on the

outer side of the joint and was as large as a bantam's egg. He complained that it kept him awake at night. X-ray (*Fig 93*) showed erosion of the outer tuberosity of the right tibia and an old stellate fracture of the patella.

OPERATION (May 25, 1932) —At the Royal Northern Hospital (E I LI). On incising the capsule a cystic mass was found to arise from the external semilunar cartilage. It measured $2\frac{1}{4}$ in in an antero-posterior direction and was 1 in wide.

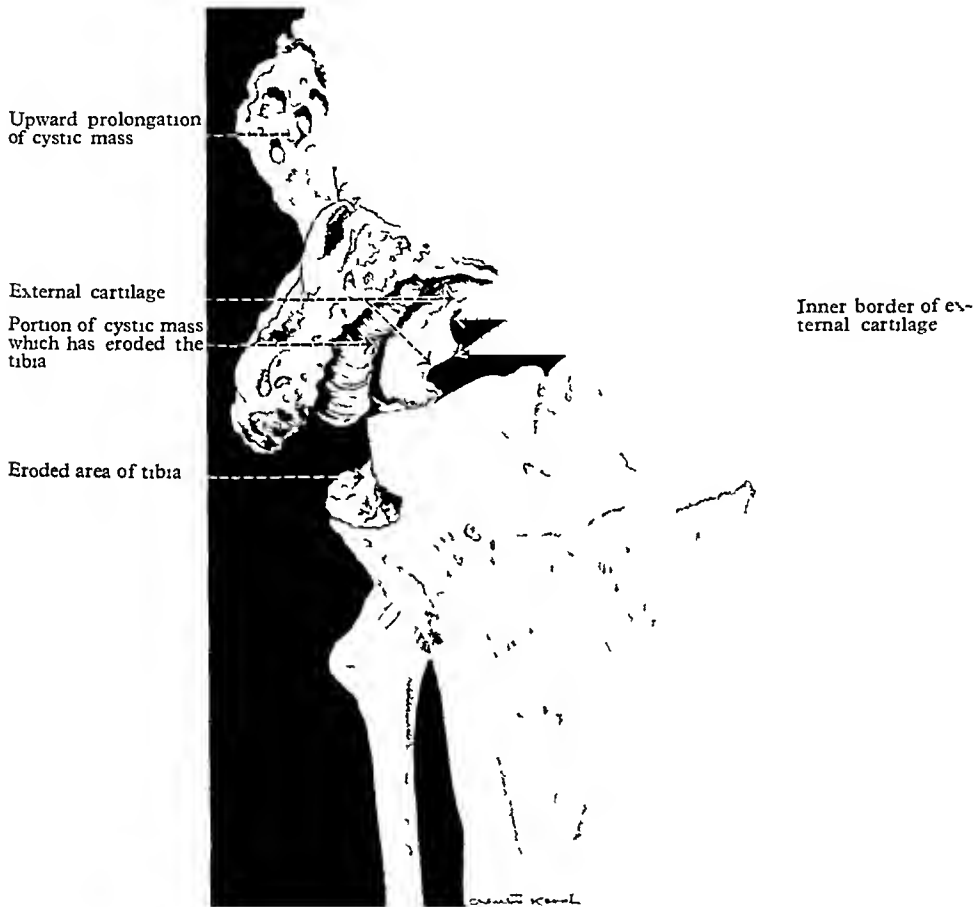


FIG 94—Case 2. Man aged 36. Composite drawing from X-ray and operative specimen. The tibia is seen from in front, and the external cartilage and cystic mass are shown as if lifted from the position which they occupied.

From its upper pole arose a process 2 in long which passed upwards in the sub-capsular tissue. The lower part of this large cyst had eroded the tibia and occupied a cavity in this bone $\frac{1}{4}$ in deep and $1\frac{1}{4}$ in long. The mass was removed with four-fifths of the external semilunar cartilage.

Mr Charles Keogh, F R C S, has kindly made a composite sketch from the X-ray and the cyst removed at operation. This clearly shows the relationship of the various parts (*Fig 94*). The cyst was multilocular and contained gelatinous material. A microphotograph shows the usual appearances of such specimens (*Fig 95*).

SUBSEQUENT HISTORY—Eight months after operation the X-ray still showed the punched-out area in the outer tuberosity of the tibia, but the patient wrote recently that he is back at his old work as a French polisher and that the knee is



FIG 95—Case 2 Microphotograph of section On the left of the field is a small cyst in the fibro-cartilage membrane It contains degenerate material and there is not a complete lining ($\times 40$)

“no disability except for a slight limp” Eight months later (i.e., a year and eight months after operation), he was walking normally and standing all day at his work

SUMMARY

Two examples of cyst of the external cartilage of the knee-joint are described in which pressure of the cyst had produced changes in the outer tuberosity of the tibia which were obvious in the X-ray Both cysts were unusually large one had been present for nearly five years and the other for ten years

ADOLESCENT KYPHOSIS

By J M EDELSTEIN, JOHANNESBURG

THE general subject of kyphosis, apart from the well-recognized type following upon tuberculous infection of the vertebral column, is one which has received insufficient attention. Though such special types as rachitic round-back of small children, the round-back of senility, and the kyphosis which is sometimes a consequence of chronic ankylosing inflammation of the column have been known for many years, inadequate recognition has been given to that antero-posterior curvature of the spine which occurs in adolescence and which is a definite clinical and pathological entity. Some such cases must in the past have been regarded as those of healed Pott's disease, and others as the consequence of the relaxed round-back of school children, which cases were in turn vaguely ascribed to postural causes, to prolonged sitting at desks, etc. The spinal deformity that is here dealt with occurs in otherwise apparently healthy young boys and occasionally in girls, has no relation as far as we know to Pott's disease, and, resulting as it does in a fixed deformity of the column, is quite distinct from the round-back which is an error of posture and which can be corrected by the patient's efforts.

It is a remarkable fact that while American and Continental literature abounds in references to this subject, it is impossible to find a description of it in the British medical literature apart from a casual few lines in one or two surgical text-books.

Scheuermann,¹ of Copenhagen, in 1921 gave a full account of the condition and made suggestions as to its etiology. It was he who first drew attention to the significance of the epiphyses above and below the bodies of the vertebræ, and named the disease 'osteochondritis deformans juvenilis dorsi', though the lesion is now usually referred to as 'Scheuermann's disease'. He suggested that under certain circumstances changes occur in the epiphysal zone somewhat analogous to those which occur at the upper epiphysis of the femur in Legg-Perthes' disease, at the tibial apophysis in Schlatter's disease, etc.

The subject of adolescent kyphosis is of considerable importance. Not only is the condition disfiguring and in some cases disabling, but its differentiation from other forms of kyphosis, especially from that due to tuberculous disease, is obviously desirable. Furthermore, there is reason to believe that, were the condition better known and recognized in its early stages, appropriate therapeutic measures might be expected not only to stay the course of the disease, but to obviate the onset and progress of the serious deformity which is its consequence.

CLINICAL MANIFESTATIONS

There is a striking difference in the incidence as between boys and girls. Of the 8 patients whose cases are here recorded, only 1 is a girl. Scheuermann's¹ cases showed a preponderance of 88½ per cent in males. Mau² described 13 cases, 3 of which were girls. Boerema,³ describing 30 cases, includes only 1 female.

It is unfortunate that most of the cases present themselves for treatment when some, if not great, deformity already exists. The subject is usually an apparently healthy boy of between 12 and 17 years. Pain in the back is not a universal symptom, though some aching on exertion is usually complained of, and occasionally there is a good deal of pain. Very often, however, the first thing to draw attention to the condition is a deformity of great or less degree which the patient's parents or friends are usually the first to notice, the patient himself being as a rule unaware of the fact that he is assuming a curvature of the back, because from the front nothing abnormal may be seen. The curvature is almost always in the dorsal region of the spine, though some authors^{4, 5} have described a lumbar spine involvement analogous to that in the thoracic region. The musculature of the back is uniformly excellent and the general health surprisingly good.

The curvature of the column is evident enough on examination (*see Figs 100, 101*), and in addition to the kyphos there is sometimes a slight scoliosis. The back as a whole shows very little or no rigidity on active movement and the patient can move about freely, but the region of the kyphos is completely fixed and can be neither actively nor passively corrected, even by suspension and the use of the whole weight of the body one cannot noticeably diminish the kyphos, demonstrating that the deformity is a permanent one. The kyphos is not sharp, but gradually rounded and smooth, and is as a rule most prominent in the vicinity of the 7th to the 10th dorsal vertebræ. The ribs on each side are splayed out so that the thoracic cage acquires a dome-like character. Lumbar lordosis of more or less degree is often present to compensate for the curvature above.

In the vertebræ constituting the curved portion of the spine characteristic changes are to be seen on radiological examination. The radiographic appearances vary according to the stage of the disease, and as the interpretation of these appearances is bound up with the question of the normal ossification of the vertebral column, a brief consideration of the latter subject is necessary before the changes which follow upon the onset of the disease are discussed.

NORMAL OSSIFICATION OF THE VERTEBRÆ AND ITS RADIOGRAPHIC FEATURES

At the 10th week of embryonic life three centres appear, one in each lateral arch and one in the centre of the cartilaginous body which is to form a vertebra. After the 6th year there is a period of quiescence of ossification.

The second period of more active growth in the adolescent stage finds its anatomical expression in the appearance of new centres of ossification, and at about the time of puberty newly acquired shadows are to be seen in the radiological picture of a normal spine. Above and below each body—first in the lower dorsal region and then extending up and down the column—there appear small triangular shadows in close proximity to their antero-superior and antero-inferior corners. From these triangular formations thin linear shadows extend backwards to become slightly broadened out again posteriorly (*Figs 96, 97*). These appearances are those of the epiphysal plates—rings which are thickened peripherally and thin centrally. Before the appearance of the epiphyses the upper and lower surfaces of the body are convex, the plates are biconcave, and between them and the body are the transparent epiphysal cartilages. With this normal state of affairs the

intervertebral spaces are of course clear and the vertebral bodies are sharp and distinct in outline. It is only in a lateral view of the spine that the epiphyses are clearly seen, for in an antero-posterior view they are overshadowed by the articulating processes, etc.

These vertebral body epiphyses have been known for a long time, but their significance has not always been apparent, and as recently as 1920 the small triangular pieces at the anterior margins of the body were referred to in an important publication⁶ as tuberculous sequestra.



FIG 96—X-ray photograph of the spine of a boy of 15 illustrating the normal appearances of the epiphysal plates above and below the vertebral bodies, as seen in lateral view

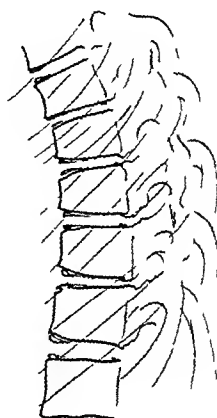


FIG 97—Diagrammatic representation of a normal spine at the age of 15, showing the epiphysal plates

Date of Appearance of the Epiphyses.—The question of the date of the first appearance of these epiphyses is an important one and has been the subject of considerable debate. Gray⁷ gives the age as 16 years. Scheuermann¹ says that 14 to 15 is the age of their first appearance. Mau² observes a difference in age of appearance according to sex, and gives the age for girls as 13 to 14 and for boys as 15 years. Personally, I have seen such epiphyses, or at any rate traces of such epiphysal formations, in skiagrams of several normal spines at as early an age as 12 to 13 years. This practically corresponds to the observations of Buchman,⁸ who noted such epiphyses in spines of subjects as young as 11½ years. There seems to be an individual variability, but I believe that some evidence of the newly developing centres can be observed in good X-ray negatives of most children of about 12 years of age.

Union of the Epiphyses with the Body.—The epiphyses unite with the vertebral bodies between the ages of 22 and 24 years, first in the cervical region, and last in the lumbar, and up to the latter age some sign of their partial or complete separation from the bodies is usually observed.

Function of the Epiphyses.—It is usually assumed that the epiphysal plates contribute to the growth in height and form of the vertebral body. Schmorl,⁹ however, from his extensive studies of the development of the spinal column, has

denied that these are in fact growth epiphyses—a point of view which he utilizes in expounding an entirely different conception of the etiology of the disease from that which is usually accepted and to which further reference will be made

RADIOGRAPHIC CHANGES IN ADOLESCENT KYPHOSIS

Having discussed the ossification and the corresponding radiographic appearances of the normal spine at about the age of puberty, we may proceed to a consideration of the radiographic aspects of the affection. The pathological changes necessarily vary with the stage at which involvement of the spine is seen. These changes can be observed very clearly in a lateral view, and, when taken in the early phases of the disease, such an X-ray picture shows the epiphysial plates—especially anteriorly—rarefied, irregular, with their outlines frayed, and not sharp and triangular as is normal. The adjacent vertebral bodies show indistinctness of outline above and below, suggesting some abnormal process at the line of growth (*Figs 98, 99*). This process is more marked anteriorly than posteriorly, with the result that the anterior surfaces of the vertebræ become shorter than the posterior, and even in the early stages some wedge-shaped deformation of the bodies is seen. The intervertebral spaces between the plates are no longer clear but cloudy and usually diminished in width.



FIG 98—Diagrammatic representation of radiographic changes seen in an early case of adolescent kyphosis (*see Case 1*)

At a further stage there may appear actual fragmentation of the epiphysial plates with varying densities of the fragments. An appearance as of a 'field of ruins' is often seen, and it is difficult to decide whether these are derived from the body or from the bony epiphysis (*see Fig 99*). The 'remains' appear as though pressed into the bodies, the latter showing more or less atrophy, especially at the apex of the kyphos.

The deforming and destroying phases may proceed very rapidly. As a rule, however, the process is of a very gradual nature and takes many years, during which time the deformity becomes progressively worse. As the disease runs its course, more distinctness becomes evident in the epiphysial plates, and they may even show a density above the normal. This is the reparative stage, which sets in gradually during the years in which the epiphysial line normally consolidates. There is a return of differentiation of the body outlines and their irregular contours become sharper and clearer. The onset of the third stage varies within fairly wide limits, which correspond to the variability of fusion time of the epiphyses, and, as in normal fusion, repair commences at the anterior parts of the body. With the conclusion of the growing age, the process of ossification being completed, the progress of the deformity is checked, but the wedging remains permanently, and, in the final adult picture of a case of adolescent kyphos, a dense appearance of the superior and inferior surfaces of the wedge-shaped bodies becomes evident.

The anterior corners of the affected bodies often take on a spur-like shape in the later stages—even one of my early cases showed this—and an appearance as of buttressing and bridging of the spondylitis deformans type becomes, in many cases, radiologically evident in the course of years.

The appearances above described are usually confined to two, three, or four vertebræ, most often the 7th to 10th dorsal being affected. The final deformation of the vertebral bodies varies with the severity of the initial process, and so gradations are seen of slight, medium, and severe wedging of the affected vertebræ, with corresponding degrees of spinal curvature.

Radiographically, the disease can therefore be traced from its beginnings in the years of puberty, continuing through the whole adolescent period, and only terminating at the time of cessation of normal growth.

CASE REPORTS

Case 1—F C B, schoolgirl, aged 14. For the past four months she had complained of some backache. The mother had noticed the child becoming increasingly round-shouldered during the last six months.

ON EXAMINATION—Patient in robust health, more than the average height for her age. The back shows moderate kyphos in the lower thoracic region, there is slight tenderness on pressure over the spinous processes of 7th, 8th, and 9th dorsal vertebræ, no rigidity of the spine as a whole, but hyperflexion and hyperextension give rise to a little pain, musculature of back good. Nothing unusual in the past history, internal organs normal, kyphos is partially corrected on suspension.

Radiological Examination (Fig 99)—Shows three dorsal vertebræ affected by a process typical of the earlier stages of the disease. This is best seen between the 8th and 9th, and 9th and 10th vertebræ. Here, in contrast to the clarity of the epiphysal plates of the vertebræ above, there is seen a hazy outline of the epiphyses and a blurring of the intervertebral spaces. The outlines of the inferior borders of the 8th, 9th, and 10th vertebræ are irregular, whilst in the inferior border of the 9th there appears to be a loss of substance, a small irregular piece remaining, however, which seems to belong to the vertebral body but is separated from it by a clear space. The 8th, 9th, and 10th vertebræ are already slightly but definitely wedge-shaped, and, what is very remarkable in a case of such short duration, the 10th vertebra appears saddle-shaped anteriorly with the antero-superior margin presenting a spur-like formation.



FIG 99—*Case 1*. X-ray illustrating characteristic appearances of an early case. Note the fragmentation of the epiphyses. Those of the 7th vertebra are unaffected, but the appearances at its lower border suggest the possible presence of nuclear hernia.

Case 2—J M, schoolboy, aged 14. Healthy, athletic, perfectly straight, until very severe attack of bronchopneumonia, after two months was discharged fully recovered, except for curvature of back noticed by nurses when he first got up, had never complained of pain in his back. Was treated by massage and exercises, but eventually was sent for investigation because Pott's disease was suspected, the deformity having become progressively worse. Complains now of backache on exertion.

ON EXAMINATION—Patient is well-built but for marked curvature of the thoracic spine which embraces a higher level than usual, the curve appears to commence about the 4th dorsal vertebra, sloping gradually to its greatest prominence at the level of the 7th. Shoulders drooping, chest very flat anteriorly (Fig 100), scapulae very prominent, slight deviation

of spine to the left, most marked at 8th dorsal vertebra, whole back markedly arched. No lumbar lordosis, but the neck is forced forward, carrying the head prominently in front of the line of the body (Fig 101). Cervical and shoulder movements normal, region of kyphos fixed and unalterable. No muscle rigidity. Movements of flexion, extension, and side bending free.



FIG 100—Case 2. Photograph of patient showing flattening of chest as seen from the front.



FIG 101—Case 2. Photograph of patient showing lateral view of deformity.

Radiological Examination—Shows considerable blurring of the intervertebral spaces between the 6th and 7th, and 7th and 8th dorsal vertebrae. Some fragmentation of the epiphyseal plates is seen anteriorly. Wedging of the vertebrae is a marked feature, whilst the borders show decalcification.

The case is of considerable interest in view of the history of onset either during or very soon after the attack of bronchopneumonia.

Case 3—C A, schoolboy, aged 15. Four months ago the parents noticed that he was becoming round-shouldered, has had no pain, but has lately felt disinclined to take part in sport, had been diagnosed by his doctor as a postural defect, but he is unable fully to correct the deformity. No previous illnesses.

ON EXAMINATION—Well-built boy of normal height and weight, marked kyphos in lower dorsal area extending from 7th to 12th dorsal vertebrae. The curvature is to some extent, but by no means fully, correctable, hyperextension free, slight limitation of lateral movements and of flexion, compensatory lumbar lordosis, slight scoliosis, left lower ribs more prominent than right, slight tenderness on pressure over vertebral spines at kyphos.

Radiological Examination (Fig 102)—Shows general blurring of the shadows of all the lower dorsal epiphyses, in fact, the anterior parts are practically unrecognizable. The margins of the vertebral bodies are everywhere uneven, and the contiguous surfaces of the 10th and 11th show considerable excavation anteriorly, with no trace whatever of the epiphyses, the intervertebral space between the 8th and the 9th is markedly narrowed. Appearances are suggestive of an extensive derangement of ossification.

Case 4—D S, male undergraduate, aged 17. Developed "a cold" accompanied by pain in the side of the chest, got up after three days, but continued to have intermittent pain in the chest. He noticed at this stage that he was becoming round-shouldered, doctor's

examination revealed a spinal curvature, and, Pott's disease being suspected, he was sent to Switzerland for six months, has been wearing a spinal support since his return. Still complains of occasional ache in the right side of the chest, especially when he leans back, no pain in the back except when he tries to straighten himself or walks much without his spinal support.

ON EXAMINATION—Patient robust, height 6 ft. Generalized dorsal curvature with maximum convexity at 8th and 9th vertebræ, slight scoliosis to left, most marked at 5th and 6th dorsal vertebræ, with slight compensatory curves to the right above and below. Front of chest not flattened, chest expansion good, musculature of back excellent, no limitation of movement.



FIG 102—Case 3. X-ray illustrating early stage. The epiphyses are practically invisible and body margins very irregular.



FIG 103—Case 4. X-ray illustrating involvement of five vertebræ with great deformation of their bodies and fragmentation of their epiphyses.

Radiological Examination (Fig 103)—As many as five vertebræ are seen to be affected, viz., 6th to 10th dorsal. There are marked irregularities and variations in density at the superior and inferior surfaces of these vertebræ, especially of the 6th and 7th. Considerable fragmentation of the epiphyses is evident. The intervertebral spaces between the 6th and 7th, and 7th and 8th vertebræ are greatly diminished, and the corresponding bodies show much deformation.

Case 5—T. H. S., male, chemist's assistant, aged 17. Left school and started work at age of 14½. Friends commented on his becoming round-shouldered, and since then has been getting gradually worse, has never felt pain or the slightest discomfort, no past illnesses except bronchitis in infancy, no history of injury.

ON EXAMINATION—Patient is rather thin, but otherwise of good build, height 5 ft 6 in. A gradual extensive curvature in the thoracic region with its greatest prominence at 8th and 9th dorsal vertebræ, thorax dome-shaped, scapulæ with lateral and posterior parts of thoracic cage projected prominently backwards. Musculature of back extremely well

developed, some lumbar lordosis present, flexion and side-to-side movement normal, no muscle spasm, hyperextension at the lumbar spine easily performed. Region of kyphosis completely immobile and resists all attempts at correction.

Radiological Examination (Fig 104)—The epiphysal plates above and below each vertebral body are clear and triangular in the upper part of picture, but those between the 7th and 8th, and especially between the 8th and 9th, show blurring of their shadows. The plates appear to be compressed and flattened out. The interval between the 9th and 10th thoracic vertebrae is of interest because while no epiphyses can be seen here, the whole of the upper margin of the 10th shows serration. The 7th and 8th bodies are wedge-shaped.



FIG 104 —Case 5 Illustrating contrast between normal and affected epiphyses, the latter showing increasing degree of involvement from above downwards



FIG 105 —Case 6 X-ray illustrating marked wedge formation of vertebrae and spur like appearances at anterior margins

Case 6—W H M, shoemaker, aged 19. Since the age of 14 he was often upbraided by his schoolmaster for not standing upright. Found it impossible to hold himself erect for more than an hour, and gradually became more and more round-shouldered. Never had pain, but sought advice only in view of his ungainly appearance, not handicapped in any way, plays football and other games without difficulty.

ON EXAMINATION—Excellent physique, height 6 ft. Marked rounded kyphosis, with whole thorax domed backwards and prominence of 7th, 8th, and 9th dorsal vertebrae, marked lordosis, also pronounced forward position of upper dorsal and cervical vertebrae, movements normal except flexion, which, though good, does not enable him to touch his toes.

Radiological Examination (Fig 105)—Shows considerable wedging of the 8th, 9th, and 10th thoracic vertebrae, the superior and inferior margins of which present an appearance as though the epiphyses, misshapen but still visible, had been pressed into the bodies anteriorly. The upper border of the 10th vertebra shows considerable excavation. A notable feature is the spur-like formation of the vertebral margins anteriorly.

Case 7—N C, bricklayer's assistant, aged 18. At the age of 14 his father noticed that his back was deformed, family doctor made a diagnosis of "consumption of the spine", was sent to convalescent home for some months, more recently has undergone gymnastic exercises and massage without effect.

ON EXAMINATION—Rather thin and anæmic, height 4 ft 10 in, has no pain, but is much concerned by the deformity. Marked kyphos in lower dorsal region, most prominent at 7th, 8th, and 9th dorsal vertebræ, thoracic cage very prominent on either side, some compensatory lordosis, chest expansion poor. Movements of back remarkably good, can easily touch toes, region of deformity quite rigid.

Radiological Examination—Shows considerable wedging of the 8th, 9th, and 10th dorsal vertebræ, there is some irregularity and increased density of their superior and inferior borders. No epiphyses are to be seen, and the appearances suggest the healed stage of the disease.

Case 8—C. G., baker's assistant, aged 24. Went to doctor a year ago because of pain in the back, has never had pain before, nor has he worried about his spinal deformity, of which he has been aware for the past seven years, has always been athletic and has only been hampered in his duties recently because of pain.

ON EXAMINATION—Muscular and well-built, except for moderate curvature in region of 8th, 9th, and 10th dorsal vertebræ. Movements slightly limited, kyphos is fixed, slight lordosis, some tenderness over kyphos, wears spinal support, which relieves his pain.

Radiological Examination (Fig 106)—Shows wedging of 8th, 9th, and 10th dorsal vertebræ, their margins are reconstituted but for slight irregularities and variations in density, the intervertebral spaces are diminished, the epiphyses are united, there is no spur-like formation. The pain at present complained of suggests commencing arthritis.



FIG 106—*Case 8* X-ray illustrating healed stage, with more or less reconstituted vertebral margins.

ETIOLOGICAL CONSIDERATIONS

The question of the etiology of adolescent kyphosis, though of the greatest interest, is shrouded in much obscurity.

Is the deformity the consequence of interference with the normal development of those structures responsible for the proper growth in height and shape of the vertebræ, or is the effect on these structures merely secondary to a kyphotic deformity from other causes?

Change of structure in the early or florid stage is obviously not simply the result of compression, because change of structure can be seen to precede change of form. Buchman⁸ mentions two cases without spinal deformity which showed a radiographic involvement of the spine similar to other cases with deformity typical of the disease—a discovery which convinced him that the epiphysial involvement is primary.

Derangement of Epiphysial Development.—From many points of view the theory of an interference with the normal development of the epiphysial plates as a causative factor has much in its favour, though what is responsible for such interference is far from clear. Histological sections from cases of actual disease are of course not available, as the condition is non-fatal. There can be said to be as little agreement as to the cause of this interference in the case of the spine as there is regarding the other epiphysial disturbances such as

characterize Legg-Perthes' disease of the hip, Kohler's disease of the tarsal scaphoid, etc

On the assumption of a defect in epiphysial development being responsible for the supervening kyphosis, the latter would further disturb, by pressure, the growth which has already been delayed by the original disease. A vicious circle thus occurs which ceases at the moment when re-calcification supervenes in spite of all, and the deviation becomes fixed.

Theory of Infection—There is no clinical justification for believing that the condition is an inflammatory lesion brought about by infection. Axhausen's suggestion in regard to the etiology of other epiphysial affections—that emboli of avirulent organisms interfere with the circulation and cause a necrosis in the epiphysial areas—if true at all in such cases, can hardly apply to the spine, as the process extends to several vertebræ at the same time. It is far-fetched to assume that several epiphysial rings should fall victims to an infection at the identical time.

Theory of Increased Load and Diminished Capacity—The onset of the disease at the age of puberty, and especially in boys, when the body framework is called upon to withstand the demands of greater activity at a period of active bone generation, suggests the possibility of mechanical strain on such actively growing areas as a factor in the etiology. Mau² especially stresses the importance of mechanical strain on, and diminished capacity of, the actively growing spine.

1 *Functional Trauma*—Most writers on the subject emphasize the special incidence of the disease in agricultural labourers and in others who are subjected to hard manual labour, excessive lifting, etc. It is suggested that an increased static demand may result in marked changes at the line of growth of those vertebral bodies which mechanically are subject to greatest strain, and it is certainly true that the 8th, 9th, and 10th vertebræ might be especially vulnerable in that regard, since, owing to the normal thoracic convexity, they are situated far behind the line of gravity. In his most recent article on the subject Scheuermann¹⁰ gives it as his opinion that "the ossifying process is affected either by an isolated severe traumatism or by a series of occasional traumatic lesions. There is then a temporary overloading which produces in the anterior parts of the vertebræ, where the effect of the lesion is greatest, a temporary arrest of ossification."

In a series of experiments on rats whose tails he had kept in a state of extreme flexion by stitching them to the abdominal walls for long periods of time, Mau¹¹ observed alterations in the epiphysial cartilages amounting to atrophy and cessation of endochondral bone formation. It is debatable whether one can apply to the disease under discussion, in adolescent human beings, such a conception based on extreme compression of vertebræ in rats.

In none of my cases has there been special evidence of undue occupational strain, and I doubt whether this factor plays the all-important role ascribed to it. All the cases under my observation had a very well-developed musculature, and Case 2 is particularly instructive in this connection, as the deformity was definitely first observed after a lengthy period of recumbency.

2 *Diminished Capacity of the Growing Spine*—This is a factor not easy to evaluate. Whilst it is true that during the period of active growth the strength of bone is diminished, we do not see in *normal* growing bone any inability on the part of the tissues to adapt themselves to increased functional requirements. It is therefore doubtful whether the physiological weakness which goes with rapid

growth is a very important cause of diminishing the capacity of the column to bear weight. Nor can so-called 'late rickets' as a weakening factor be incriminated, since, apart from the fact that there are no other deformities present, recent researches¹² into the blood chemistry of the various types of the osteochondritides, which included 19 cases of vertebral epiphysitis, have shown that the calcium and phosphorous contents of the blood serum fall within normal limits.

Schmorl's Theory of Nuclear Hernia.—The supposition that the *primary* cause of adolescent kyphosis is some disturbance of epiphysial development is seriously challenged by Schmorl¹³⁻¹⁵. According to him, the views hitherto held on the function of the epiphysial plates are erroneous—that rather than being concerned with the growth of the vertebral body itself, they merely form, after cessation of endochondral growth, the circumferential edge for fixation of the intervertebral discs. The vertebral bodies, covered on their intervertebral aspects by cartilaginous tissue—the cartilage plates—develop like short bones from the subchondral zone. The cartilage plates are set directly on the spongy cancellous tissue at the upper and lower aspects of the vertebral body. The two structures are called upon to withstand the pressure exerted by the nucleus pulposus of the intervertebral disc, which is confined between the two cartilage-covered vertebræ. There is no layer of compact bone, and even the cartilage plates are present only over the central parts of the body. It is to the peculiar elastic properties of the nucleus pulposus of the disc that we must look, according to Schmorl, for an explanation of disturbances in the osteogenetic zone of the vertebral body. The rôle presumably played by the nucleus pulposus in the etiology of spinal deformity and disease has received a great deal of attention, and Beadle¹⁶ has recently published an extensive account of the researches on the subject.

Prolapse of the nucleus pulposus may, according to Schmorl, occur as the result of congenital defect of the cartilage plate. Such possible disturbances of development were suggested by his examination of 4000 spines amongst which he found, in quite young children, evidence of nuclear prolapse in the form of "cartilaginous knots" in the spongiosa of the vertebral body. Such "cartilaginous knots" are regarded as lying within the range of normality and are said to assume a pathological character only when, under the influence of great functional demand, there occur other changes, such as further tearings of the cartilage plate with further herniation of nuclear tissue into the spongiosa.

Having demonstrated such nuclear prolapses even in children, Schmorl assumes a congenital predisposition to the development of adolescent kyphosis. The deformity occurs only during adolescence because, from overloading of a potentially weakened spine, a disproportion exists between the resistance of the intervertebral plates and the weight. Trauma pure and simple, without the presence of congenital defect, may also result in herniation of the nucleus pulposus by tearing a normal cartilage plate.

The pathological sequence of such nuclear prolapse is conceived as follows. Where, with overloading or trauma, there is nuclear prolapse into the spongiosa, there the process of ossification in the *subchondral zone* is distributed, so that there is an interference with the growth in length of the vertebral body, since such growth depends upon proliferation of the spongy tissue of the body itself. As a consequence of the prolapse of the disc, however, there is a reactive new formation of bone, and, near the upper and lower surfaces of the body, irregularities of

ossification occur which account for the serrated margins of the body and for the presence of areas of condensed bone around the periphery, both of which can be seen radiologically. This disturbance holds back the growth of the epiphysal ring and may even distort it, but the marginal irregularities, according to Schmorl, are not due to an epiphysal involvement as such, but to the effect on the *bodies* of the prolapse of the nucleus pulposus.

There has been considerable support for the views of Schmorl on the etiology of adolescent kyphosis, though they are based entirely on post-mortem evidence and not at all on clinical data. It is true that the inferences are drawn from a series of spines in not one of which was there post-mortem evidence of epiphysal disturbance, and that the series included six young subjects with kyphosis, who had died between the ages of 16 and 24. The dates of development of the kyphoses are, however, not given, as clinical histories were not available. It is thus quite uncertain whether the six cases of kyphosis referred to by Schmorl belonged to the category of 'adolescent kyphosis'. Furthermore, reconstitution of affected epiphyses after the subsidence of the early stages of the disease is not only a possibility, but is definitely suggested by the radiological appearances in the late stages.

There are, indeed, many clinical and radiological aspects of adolescent kyphosis which remain unexplained by Schmorl's theories. The characteristic blurred outlines of the *anterior* parts of the vertebræ in the 'florid' stage of the disease are difficult to explain on such a conception, especially in those cases with comparatively rapid onset of the kyphosis. It is also not clear how the typical cycle of changes lasting over several years can be regarded as a process in which the epiphyses play but a secondary part. Neither is that part of the theory which presupposes a traumatic factor borne out by the histories of the cases under my observation.

As to the radiological appearances, one would expect that evidence of such nuclear prolapse would be forthcoming in all cases of adolescent kyphosis. With the possible exception of *Case 1*, where the appearance of the *unaffected* body of the 7th vertebra is suggestive (*see Fig 99*), I have failed to satisfy myself that such exists, either in my own comparatively small series of cases or in that of a larger group of X-ray negatives typical of the condition which I recently had the opportunity of examining at the Clinic of Professor Haas in Vienna.

It is of interest in this connection that Calve and Galland,¹⁷ who subscribe to Schmorl's views, yet find it necessary to invoke a purely epiphysal lesion independent of nuclear prolapse as an explanation for at least 3 of their series of 26 cases. A remarkable case is recorded by them which, on first observation at the age of 15 years, showed a kyphosis with "epiphysitis and sub-epiphysal rarefaction", and which *two years later* showed radiological evidence of "very small nuclear hernias". Six years later, while the epiphyses were united and the vertebræ wedge-shaped, disc hernias were found to be numerous and prominent.

That nuclear prolapse occurs cannot be denied, many radiological observations of such lesions having been reported, but whether such prolapses have any bearing on the etiology of adolescent kyphosis is by no means proven. Radiological evidence of nuclear prolapse in cases of such deformity may be purely incidental.

Other Suggested Causes—That the deformity has anything to do with muscular asthenia can be dismissed by the fact that the muscle development is always good.

Endocrinal disturbance affecting normal bone growth has been suggested here as in other epiphysial derangements, but there is at least no naked-eye evidence to justify such an assumption

The idea of circulatory disturbances at the line of growth from functional trauma, leading to vasospasm, ischæmia, and aseptic necrosis, fails to explain why the epiphyses of some children are so much more vulnerable than others

CONCLUSIONS AS TO ETIOLOGY

If one discards the congenital basis of Schmorl's conception of the etiology of adolescent kyphosis, one is forced to the conclusion that a developmental disturbance arises *de novo* during the years of puberty. What the nature of that disturbance is, and what its immediate cause, awaits elucidation, but no doubt the question is bound up with the general problem of the etiology of osteochondritis in young subjects. It is possible that a nutritional defect, inimical to normal epiphysial development, will prove to be the key to the problem of disturbances in the vertebral column as well as to those in other areas of epiphysial growth. We have in rickets an example of an error of assimilation, or of nutrition, or of both, which affects growing bone so profoundly that distortion comes with ordinary weight-bearing. There are many other biochemical factors besides those of the assimilation of calcium and phosphorus about which, in relation to growing bone, we understand too little.

One cannot but feel that all the etiological possibilities previously discussed are in the final analysis at most of secondary importance only, and that one has to seek for some hitherto undetermined biochemical disturbance to explain why the epiphyses of some children fall victims to circumstances which in others have no effect. It is probable that the only significance of the much-vaunted factor of "multiple small traumata" is that of a localizing or, at most, an aggravating agency.

In connection with the general subject of epiphysial growth disturbance in children, another lesion of the spine which occurs in the 2 to 7 year age-period may be mentioned. Described by Calve,^{18, 19} under the name of "osteochondritis of the vertebral body", it bears certain analogies to the disease which is the subject of this article, and results in an interference with the development of the *primary* epiphysis for the body centrum. The affection, one example of which has come to my notice, leads to flattening and sclerosis of the affected vertebra and to the development of a kyphos. The analogy between this disease and adolescent kyphosis is emphasized by the fact that in both cases the age of onset is the period of most active growth for the area involved. May there not be also some analogous nutritional factor in both diseases?

TREATMENT

It is not an easy matter to formulate the treatment of a condition the etiology of which is still so obscure. It is, however, necessary to stress the importance of early recognition of the disease. The onset of even the slightest kyphosis, or of what is popularly known as 'round shoulders', in a subject at the age of puberty should demand a radiological examination at the earliest possible moment. Prophylactic measures in the early stages should do much to minimize deformity. It is

essential, as was carried out in *Cases 1 and 2*, that in such comparatively early cases complete recumbency should be adopted in order that weight-bearing on the affected column may be reduced to a minimum. In recumbency the ventral parts of the whole column are pulled apart and further compression of the bodies and their epiphyses is lessened. This effect can be heightened if a suitable frame is provided to keep the spine hyperextended. Traction to the legs and head, with elevation of the upper end of the bed, is of great advantage. After a minimum period of three months on the frame, and longer if radiological control demands it, the patient may be let up in a plaster-of-Paris jacket or a leather support. The latter appliance, worn for several years after, is of value in preventing the increase of residual deformity, which unfortunately occurs to some extent, in spite of most careful treatment. Pain is most certainly relieved by the wearing of a posterior support.

It is possible that earlier radiological examination of youths who complain of 'backache' may reveal the condition before the appearance of kyphosis. This, of course, is not a simple matter because so many of the cases have few or no symptoms in the early stages.

It is very doubtful whether for those cases with deformity of considerable duration much can be accomplished. Mobilizing measures such as gymnastic exercises, massage, etc., are of negligible value at a stage when fixed rigidity has been present during several years. One's own experience is that orthopaedic gymnastics have not the slightest effect on the deformity in the very late cases, and it is of course obvious that in the earlier stages such measures are definitely contra-indicated. There are, however, intermediate stages where the deformity has not yet reached its maximum and where measures to prevent the progress of such deformity are called for. Plaster-of-Paris jackets, put on in as much hyperextension of the spine as it is possible to obtain, are to be applied and re-applied at intervals, until such time as the deformity shows no tendency to progress. As a precautionary measure a posterior spinal support may be used later.

Treatment on the whole cannot be said to be satisfactory in its results, and that is perhaps to be expected, in view of the present state of our knowledge with regard to the etiology of the lesion which is responsible for the deformity.

SUMMARY

1 A kyphotic deformity may occur in adolescents resulting from disturbances of vertebral ossification.

2 The ossification of the column and the deviations from the normal consequent upon the onset of the growth disturbances are described.

3 The lesion results in wedge-shaped deformation of several vertebral bodies. The kyphosis which this gives rise to, being sometimes unaccompanied by pain, may at first pass unnoticed. Movements of the spine are but little affected, but disfigurement is progressive.

4 Eight cases are reported illustrating various phases of the disease, and their radiological features are described.

5 A critical survey of the etiology is presented and the conclusion is drawn that most, if not all, the etiological factors usually cited to explain this and other epiphyseal disturbances of a similar nature are of secondary significance only.

6 The tentative suggestion is made that some biochemical disturbance, based possibly on an error of nutrition or of assimilation, is at the root of such epiphyseal affections

7 The lines of treatment are indicated and special emphasis is laid on the importance of early recognition of the disease

I desire to acknowledge my indebtedness to the orthopædic surgeons of Liverpool for permission to include some of their cases in the group which is the basis of this article

REFERENCES

- ¹ SCHEUERMANN, H, "Kyphosis dorsalis juvenilis", *Zeits f orthop Chir*, 1921, xli, 4, 305
- ² MAU, C, "Die Kyphosis dorsalis Adolescentium im Rahmen der Epiphysen und Epiphysenlimerkrankungen des Wachstumsalters", *Ibid*, 1924-5, xlv, 145
- ³ BOEREMA, I, "Ueber Kyphosis dorsalis Adolescentium", *Arch f klin Chir*, 1931, clvi, 737
- ⁴ ECKARDT, F, "Ueber das klinische Bild der Scheurmannschen Krankheit", *Arch f Kinderheilk*, 1932-3, xcvi, 81
- ⁵ LINDEMANN, K, "Die lumbale Kyphose im Adoleszentenalter", *Zeits f orthop Chir*, 1932, lvi, 54
- ⁶ LEHRENBECKER, A, "Ueber seitliche Wirbelaufnahme bei Spondylitis tuberculosa", *Forts a d Geb d Rontgenstrahlen*, 1919-20, xxvii, 643
- ⁷ GRAY, H, *Anatomy Descriptive and Applied*, 25th ed, 1932 London Longmans Green & Co
- ⁸ BUCHMAN, J, "Vertebral Epiphysitis A Cause of Spinal Deformity", *Jour Bone and Joint Surg*, 1925, vii, 814
- ⁹ SCHMORL, G, "Zur pathologischen Anatomie der Wirbelsaule", *Klin Woch*, 1929, viii, 1243
- ¹⁰ SCHEUERMANN, H, "Cyphose juvenile", *Arch med belges*, 1928, lxxxi, 353
- ¹¹ MAU, C, "Tierexperimentelle Studien zur Frage der pathologischen Anatomie der Adoleszentenkyphose", *Zeits f orthop Chir*, 1929, li, 106
- ¹² BUCHMAN, J, and GITTLEMAN, I, "Inorganic Blood Chemistry in the Osteochondritides", *Amer Jour Dis Child*, 1930, xl, 1250
- ¹³ SCHMORL, G, "Die Pathogenese der juvenilen Kyphose", *Forts a d Geb d Rontgenstrahlen*, 1930, xli, 359
- ¹⁴ SCHMORL, G, "Ueber die pathologische Anatomie der Wirbelbandscheiben", *Betr z klin Chir*, 1931, cli, 360
- ¹⁵ SCHMORL, G, "Ueber Knorpelknoten an der Hinterfläche der Wirbelbandscheiben", *Forts a d Geb d Rontgenstrahlen*, 1929, xl, 629
- ¹⁶ BEADLE, O A, "The Intervertebral Discs Observations on their Normal and Morbid Anatomy in Relation to Certain Spinal Deformities", *Med Research Council, Special Report Series*, No 161 London 1931
- ¹⁷ CALVE, J, and GALLAND, M, "Clinical Aspects of 24 Cases of Vertebral Nuclear Hernia and 3 Cases of Epiphysitis", *Rev d'Orthop*, 1930, xvii, 723
- ¹⁸ CALVE, J, "A Localized Affection of the Spine Suggesting Osteochondritis of the Vertebral Body, with the Clinical Aspect of Pott's Disease", *Jour Bone and Joint Surg*, 1925, vii, 41
- ¹⁹ CALVE, J, "Chondrite vertebrale infantile", *Robert Jones Birthday Volume*, 1928 Oxford University Press

ENTEROGENOUS CYSTS

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THE following are the notes of a patient who was under the care of Mr A E Coates at the Melbourne Hospital

A McA, male, aged 32 years For many years there had been pain about two hours after meals, intermittent attacks of vomiting, each lasting for four weeks, and remissions lasting about six weeks During the attacks vomiting would occur about three times a week, without warning half-way through a meal A barium meal examination suggested the presence of a duodenal ulcer For twelve hours there had been severe epigastric pain of sudden onset Vomiting occurred once, there was much nausea and retching, and the bowels had not acted

ON EXAMINATION—The temperature was 95.6°, pulse-rate 64, and the blood-pressure, systolic 92 mm, diastolic 54 mm The abdomen was not distended, was held immobile during respiration, and there was generalized rigidity, most marked in the upper abdomen, especially on the right side Acute tenderness was elicited midway between the xiphisternum and the umbilicus, and the area of liver dullness seemed diminished A diagnosis of perforated duodenal ulcer was made

OPERATION—At operation there was no evidence of gastric or duodenal ulceration Some blood-stained fluid escaped the upper loop of the jejunum was distended, and the lower ileum was collapsed The small bowel was followed proximally till near the upper part of the jejunum there was found a strangulated cyst in the left side of the mesentery, about three-quarters of the size of a tennis ball The pedicle was ligated and the cyst removed The wall of the cyst was identical in structure with that of the adjoining small intestine

ETIOLOGY OF ENTEROGENOUS CYSTS

The view most frequently accepted is that enterogenous cysts arise from diverticula of the intestine of the embryo Another theory is that epithelium becomes detached from the embryonic intestine at a very early age and develops into a cyst Each of these conceptions will be discussed

The Theory of Origin from Diverticula.—

1 Those who believe that cysts arise from diverticula have no difficulty in demonstrating many such diverticula in the intestine of the embryo Reference is often made to the work of Lewis and Thyng, and although such diverticula as they described on the free border of the developing intestine may become isolated from the bowel and form cysts, it is unlikely that this occurs with any degree of frequency

Another possible origin is found in the irregular epithelial growth which occurs at the stage when the intestinal lumen of the embryo becomes obliterated, for small hollow stalks of cells bud into the surrounding mesenchyme and have been found cut off from the intestine in some instances

In a very careful investigation of hyperplasia of the mucous membrane of the intestine, Lauche demonstrated that sometimes intestinal glands transgressed the muscularis mucosæ of the stomach, ileum, and jejunum, and in some instances

he found in the submucosa small cysts which appeared to arise by an isolation of the glands from the intestinal mucosa Stohr, Florence, and Sabin have described similar glands in the embryos of domestic animals and of man occurring in the lymphoid follicles of the ileocæcal region

From such evidence it appears that there exist in the *embryo* numerous diverticula from which enterogenous cysts may develop It is possible that some cysts do arise in this way, but such is a matter of speculation, and complete proof is almost unattainable

2 It is very unlikely that enterogenous cysts and non-vitelline diverticula of the *adult* small intestine have a common origin, for their distribution differs so widely Diverticula occur most frequently in the jejunum, especially in its upper coils, whereas enterogenous cysts are very uncommon in the jejunum and are found in the ileum, especially near the ileocæcal junction The age incidence of diverticula and of enterogenous cysts differ widely, since the former are found in the adult and the latter usually in infancy and in childhood

3 Another theory of the development of enterogenous cysts is worthy of serious consideration Gfeller regards enterogenous cysts as arising from an unobliterated vitello-intestinal duct, either by separation of a part of the intestinal *Anlage* or by germinal displacement Edwards and Dukes think this is unquestionably the most likely hypothesis, but it seems to the author that the widespread distribution of the cysts is the greatest argument against the application of this theory to all cases There are certainly some instances where it is impossible to gainsay that a cyst in the antimesenteric border of the bowel or in the mesentery may be derived from the vitelline tract Indeed, most of the antimesenteric cysts probably arise in this way because, for reasons not understood, enterogenous cysts of non-vitelline origin are entirely situated toward the mesentery (perhaps the association of germinal displacement with blood-vessels provides the clue) But Meckel's diverticula and associated remnants are in a fairly fixed position, and the greatest departures from this position which are known to the author are, at one extreme, at the appendix (Crymble's case), and, at the other, midway in the length of the small bowel It is difficult to believe that thoracic, duodenal, and ileocæcal enterogenous cysts have their origin in the vitelline duct

The position is that enterogenous cysts appear to arise from any portion of the embryonic alimentary tract, more frequently in certain sections, and that cysts resembling enterogenous cysts in structure and characteristics arise from the vitelline tract The latter equally merit the name 'enterogenous cyst', but they must necessarily be of limited distribution

The Sequestration Theory—A very careful search is necessary in order to find evidences of the early stages of epithelial sequestration, and very few examples have been described, but this is not a bar to an etiological relationship to enterogenous cysts, since these cysts are themselves very uncommon

The author made serial sections of the intestines of a number of human embryos, and in two embryos evidence of epithelial sequestration was found Illustrations of these sections are presented (*Figs 107, 108*) *Fig 107* is from a 65-mm embryo the small cyst extended through three sections, the epithelium appeared undifferentiated, and in this resembled the epithelium of the adjoining intestine The section did not include any Wolffian remnants *Fig 108* is from an embryo of 170 mm The small cyst was lined by epithelium resembling that



FIG 107—Transverse section of the small intestine of an embryo of 65 mm. An isolated group of cells is seen at (A), and, when traced through serial sections, these cells were seen to form a closed cavity
a, Low power ($\times 37$); b, Higher power ($\times 120$)

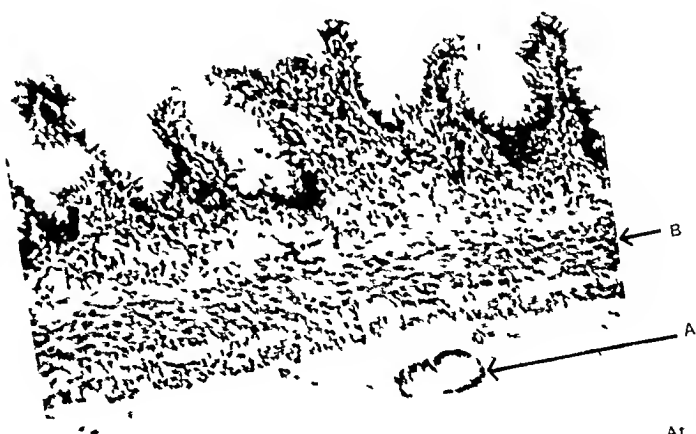


FIG 108—Transverse section of the small intestine of an embryo of 170 mm. At A is seen an island of epithelium lying outside the muscularis of the intestine (B). The epithelium resembled that of the adjoining intestine ($\times 37$)

of the bowel. Such observations prove nothing, but they demonstrate a possible source of origin for enterogenous cysts.

These small islets of epithelium must have been detached from the intestinal mucosa before the circular musculature was formed. This muscle appears in the duodenum in a 12.5-mm embryo and spreads along the intestine until it is present in the ileocaecal region in the 22.8-mm embryo. Thus the sequestration of the epithelium probably occurs before the embryo reaches the 23-mm stage. Sequestration of buds of intestinal epithelium may occur in the submucosa over a much longer period, for the muscularis mucosæ is not differentiated until the embryo measures 187 mm.

It is assumed that this isolated epithelium gains a muscle coat in the same way as the intestine by a condensation of the hitherto undifferentiated mesenchyme which surrounds the intestine.

Several workers have made observations which resemble those described except that the cysts they report were confined to the duodenum. In a pig embryo of 20 mm Lewis and Thyng described a cyst which had become detached from the intestine in the lower duodenal region. Johnson found among the duodenal villi of a seven-month embryo several small cysts which were distended with mucus and were separated from the surface epithelium by a thin layer of connective tissue.

DESCRIPTION OF THE ENTEROGENOUS CYST

The wall consists of three layers, muscular, submucous, and epithelial. The muscular layer usually consists of two strata lying at right angles to one another, but occasionally a thin single layer is found. It is this coat which causes the cyst to resemble the intestine so closely. The submucosa is a thin connective-tissue layer. Epithelium may form a complete lining, or be present in limited patches, or even be completely absent, but in this last event epithelial cells are usually found in the contents of the cyst. The epithelium forms a thin smooth layer, occasionally possesses glands, and is most frequently columnar or low cuboidal, but it may undergo transition to ciliated or stratified epithelium.

The fluid from the cyst is usually pale, clear, or straw-coloured, containing much albumin, the reaction is alkaline and the specific gravity is 1.015. The fluid is occasionally distinctly mucoid. Microscopic examination sometimes reveals blood, cell debris, and cholesterol.

The Site of the Cysts—An idea of the relative frequency in different portions of the small and large bowel is gained from a tabular summary of the 55 cases —

	CASES
Duodenum	3
Jejunum	4
Ileum (excepting last 4 in.)	16
Last 4 in. of ileum	31
Ileocaecal valve	
Ileocolic angle	
Cæcum	
First 2 in. of ascending colon	
Transverse colon	1

Of the 16 cases included as occurring in the ileum above the last 4 in., some may really belong to the lower 4 in., but an opinion could not be formed on the

published data Even so, the frequency of the incidence in the ileocaecal region is worthy of note in contrast to the rarity of the cysts in the colon beyond the first few inches of the ascending colon and their infrequent occurrence in the duodenum and jejunum The incidence in these latter organs does not appear to exceed that of enterogenous cysts in association with the trachea, œsophagus, or even the stomach

The distribution in the ileocaecal region is worthy of analysis —

Ileum within 4 in of ileocaecal valve	CASES 12
Ileocaecal valve	3
Ileocaecal and ileocolic angle	3
Cæcum	10
Ascending colon (first 2 in)	3

Position of the Cyst in Relation to the Wall of the Bowel—Details of this are mentioned or can be deduced in 45 instances The cyst may be (1) In the submucosa, (2) In the muscle layers, (3) Subperitoneal, (4) In the mesentery of the bowel, either close to or some distance from the intestine (*Figs 109, 110*)

The position of cysts in the wall of the jejunum and ileum differs from that of cysts in the ileocaecal region

In the jejunum the positions were	{ Intermuscular { Mesenteric	CASES 1 3
In the ileum (excepting the last 4 in)	{ Submucous { Intermuscular { Mesenteric	2 3 9

That is, in these cases the mesenteric distribution was predominant

In the ileocaecal region the incidence is greatest in the submucosa, and many cysts occur in the muscle layer

Ileum (last 4 in)	{ Submucous { Intermuscular { Mesenteric	CASES 5 4 1
Ileocaecal valve	{ Submucous { Subperitoneal	2 1
Cæcum	{ Submucous { Intermuscular	4 2
Ascending colon	Submucous	3
Ileocaecal and ileocolic angle	Subperitoneal	3 (1 e, mesenteric)

Another feature worthy of note is that, of the cysts in any part of the ileum, 10 were on the mesenteric side of the bowel and 5 were antimesenteric (these data were supplied only in a limited number of cases) The antimesenteric cysts are believed to originate in the vitello-intestinal duct

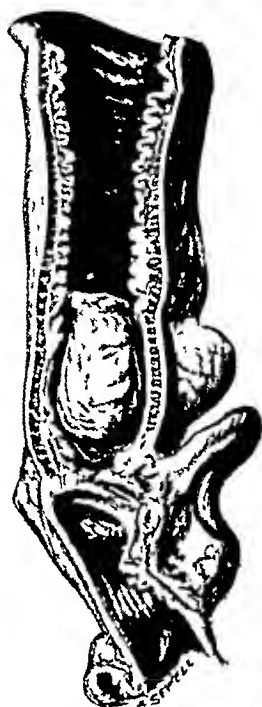


FIG 109—The terminal ileum and cæcum of a child. Immediately above the ileocaecal valve two enterogenous cysts are to be seen one in the submucosa obliterating the lumen of the intestine, and the other, deep to the serosa projecting on the surface of the ileum (*Reproduced by the courtesy of the Curator of University College Hospital Museum*)

Age when Discovered.—As a rule enterogenous cysts are found only when they cause severe mechanical disturbances in the intestine. A study of the age

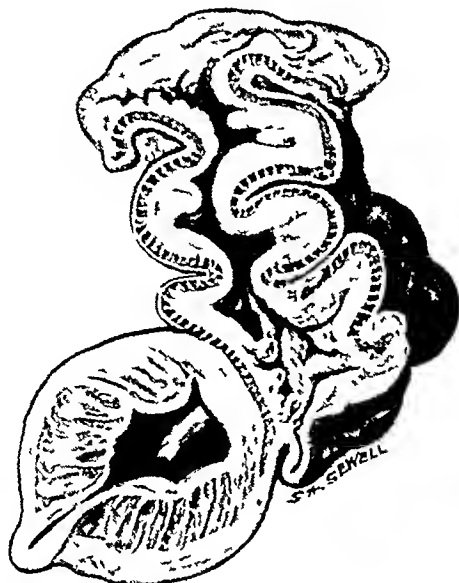


FIG 110—A specimen of great interest. Closely attached to the transverse colon is a thick-walled enterogenous cyst. A diverticulum, resembling in all respects a diverticulum of the colon, is present in the wall of the cyst. (Reproduced from St. Thomas's Hospital Museum by kind permission of the Curator.)

at which such complications occur reveals features of interest, and a brief analysis is set out —

ORGAN	AGE WHEN CYST WAS DISCOVERED
Duodenum	Close to birth
Jejunum	{ 2 in the first week of life 1 at 6 years 1 at 32 years
Ileum (excluding last 4 in)	{ 1 in a newborn child 1 at three months 8 at these years 3, 5, 7, 8, 8, 11, 14, and 62
Ileum (in last 4 in)	{ 1 in a newborn child 2 in first week of life 6 in the first year of life 2 at 5 and 10 years
Cæcum	{ 1 in a foetus of six months 3 in the first six months of life 6 at these years 5, 12, 21, 21, 23, 29
Ascending colon	{ 1 at eight months 2 at 11 and 38 years

The following facts emerge from this analysis (1) Cysts of the ileum (excluding the last 4 in) are discovered usually between the third and fourteenth years, (2) Cysts of the last 4 in of the ileum are discovered usually in the first year of life, (3) Cysts of the cæcum are discovered usually at two periods of life (a) in the first six months, (b) between twenty and thirty years.

Incidence in Relation to Sex—In 30 of the cases the sex was recorded and a slight predominance in females was noted females 18, males 12. This ratio was the same in different parts of the intestine.

CLINICAL MANIFESTATIONS

The clinical manifestations of enterogenous cysts are predominantly of a mechanical nature, and for the greater part occur as intestinal obstruction which results from stenosis, intussusception, or volvulus. An analysis of the findings at operation discloses the clinical picture which is to be expected

<i>Jejunum</i>	1 obstruction, 1 volvulus, 1 torsion of the cyst, 1 the cyst was partially torn from the bowel and general peritonitis ensued
<i>Ileum</i> (excluding the last 4 in)	4 obstruction, 2 volvulus
<i>Ileum</i> (in last 4 in)	5 intestinal obstruction, 1 intussusception
<i>Ileocaecal angle</i>	2 intestinal obstruction
<i>Ileocaecal valve</i>	2 intussusception (irreducible)
<i>Cæcum</i>	2 intestinal obstruction, 2 intussusception, 2 thought to be 'acute appendicitis'
<i>Ascending colon</i>	1 intussusception
<i>Transverse colon</i>	1 attacks of acute pain

Thus it is seen that, in 34 cases in which the symptoms were described, there was intestinal obstruction in 14, intussusception in 6, and volvulus in 3

TREATMENT

The operative treatment of cysts in the wall of the bowel is not simple, for in many instances the cyst is discovered after a search for the cause of intestinal obstruction or when an intussusception is reduced and a mass remains at the site, in the latter instance the impression is gained that the reduction is incomplete. In any case the patient is very ill and cannot tolerate any but the simplest procedures and unfortunately these often fail.

The following operations are discussed briefly (1) Enucleation of the cyst, (2) Excision of the bowel containing the cyst, (3) Marsupialization, (4) Evacuation.

Enucleation of the Cyst—This is the procedure to be adopted in cysts isolated in the mesentery, and these cases present no difficulty. Enucleation is much less successful with cysts in the bowel wall, often failing on account of a firm attachment of that portion of the cyst toward the lumen of the intestine. In some cases the cyst is removed with ease. A perusal of the cases in which enucleation was performed or attempted suggests that success was attained when the cyst was subperitoneal or intermuscular and that failure often followed attempts to enucleate a submucous cyst. It has been mentioned that an intimate attachment frequently exists between the intestinal mucous membrane and the wall of the cyst. This probably explains the failure of enucleation, and it is of interest to note that Edwards removed a cyst from the wall of the cæcum together with some of the intestinal mucosa. An extension of this procedure offers an alternative to the severe operations performed in those cases in which attempts to enucleate the cyst fail.

In collected cases enucleation was attempted in 9, and in these was successful in 5 and failed in 4. Miller, in 1913, recorded 10 cases of enucleation without a death.

Resection of Bowel—In many instances resection of the intestine containing the cyst was performed as a measure of desperation when attempted enucleation failed, and the severity of this operation in a young child was responsible for the high mortality. In this series resection was performed in 14 instances, and in only 3 cases is it stated that the patient recovered. Miller estimated that resection had a mortality of 60 per cent.

Marsupialization of the Cyst—In two instances the cyst was opened, anchored to the abdominal wall, and drained Both patients recovered, one after a second operation to excise the faecal fistula which formed through the 'pouch'

Higgins and Lloyd concluded that "enucleation was the operation of choice Marsupialization and drainage should be done in the remaining cases"

Evacuation of the Cyst—In one instance only was this performed, and death followed The danger seems to be the spread of infection from the intestine into the traumatized cyst

BIBLIOGRAPHY

- AITKEN, R Y, *Brit Jour Surg*, 1931, xviii, 521
 AYER, *Amer Jour Med Sci*, 1906, cxxxi, 89
 BALL, GIRLING, *Brit Jour Dis Child*, 1914, ii, 259
 BAZIN, *Canad Med Assoc Jour*, 1925, xv, No 2
 BECKMAN, *Ann of Surg*, 1930, xc, 1097
 BLACKADDER, *Amer Ped Soc*, 1913, xlv
 BOGERT, F VAN DER, *Jour Amer Med Assoc*, 1909, lii
 BOLTON and LAWRENCE, *Brit Med Jour*, 1916, ii, 248
 COUTTS, *Brit Jour Surg*, 1920-21, viii, 229
 CRYMBLE, *Ibid*, 1921, ix, 34
 * DITTRICH, *Zeits f Heilk*, 1886, vi, 277
 DRENNEN, *Arch of Surg*, 1931, lxii, 106
 EDWARDS, HAROLD, *Clinical Jour*, 1928, lvii, 319
 EDWARDS and DUKES, *Brit Jour Surg*, 1929-30, lviii, 7
 EVANS, ARTHUR, *Ibid*, 34
 EVE, *Med-Chir Trans*, 1897-8, lxxxix, 51
 FLORENCE, LAURA, *Amer Jour Anat*, 1922, v, 31
 FRANKEL, *Virchow's Arch*, 1882
 * GFELLER, *Deut Zeits f Chir*, 1902, 330
 HEDINGER, *Virchow's Arch*, 1904, clxxviii, 25
 * HENNIG, *Centralb f Gynakol*, 1880, iv, 398
 HIGGINS and LLOYD, *Brit Jour Surg*, 1924-25, xii, 95
 * HUETER, *Ziegler's Beitr*, 1896, xix, 391
 HUNTER, JOHN L, *Brit Med Jour*, 1922, ii, 800
 KEITH, SIR ARTHUR, *Brit Jour Surg*, 1921, viii, 454
 * KROGIUS, *Zeits f klin Med*, 1903, xlix, 53
 * KULENKAMPFF, *Centralb f Chir*, 1883, x, 679
 LAUCHE, *Virchow's Arch*, 1924, cclii
 LEWIS and THYNG, *Amer Jour Anat*, 1907-8, vii, 505
 LOTHEISSEN, *Deut Zeits f Chir*, 1925, 179
 MACAULEY, *Brit Jour Surg*, 1923, xi, 122
 MALCOLM, *Museum of Royal College of Surgeons*, 1221 I
 MILLER, ROBERT T, *Johns Hopkins Hosp Bull*, No 272, 316
 MILLER and ROBERTSON, *Brit Jour Surg*, 1929-30, xvii, 373
 * NASSE, *Langenbeck's Arch*, 1893
 * NEUPERT, *Centralb f Chir*, 1910, 714
 * PUSCHMANN, VON, *Deut Zeits f Chir*, 1904, 109
 * QUESNEL, *Nord Med Ark*, 1898, Part 6
 ROTH, *Virchow's Arch*, 1881, lxxvi
 SAINSBURY, *Trans Pathol Soc*, 1886-7, lxxviii, 146
 * SANGER and KLOPP, *Arch f Gynakol*, 1880, vi, 415
 * SELIGMANN, *Jahr Handbuch Staatshank*, 1890
 SHALLOW, *Ann of Surg*, 1925, lxxxi, 795
 SHORVON and WELLS, *Lancet*, 1932, Jan 23
 * SPRENGEL, *Verhandl d Deut Gesellsch f Chir*, 1900
 STRODE and FENNEL, *Surg Gynecol and Obst*, 1923, lxxvii, 781
 * STUDSGAARD, *Hospitaltidende*, 1894, 641
 TERRIER and LECENE, *Rev de Chir*, 1904, lvi, 161
 TURNER and TIPPING, *Proc Roy Soc Med*, 1913-4, vii, 29
 WAUGH, OLIVER, *Surg Gynecol and Obst*, 1923, lxxvii, 785

THE ORIGINS AND EVOLUTION OF COLOSTOMY

BY TILSON DINNICK, LONDON

WOUNDS of the bowels have been recognized from remote antiquity. Perhaps the earliest reference is to be found in the Book of Judges, where we read how Ehud, a left-handed man, stabbed Eglon the King of Moab—"and his dirt ran out and he died." Hippocrates regarded all such wounds as 'deadly', and even Celsus advised that their cure be left to Nature.

It could not, however, have escaped the eye of some more acute observers that sometimes these wounded patients survived, Nature establishing a faecal fistula. Others noticed that Nature herself in some of her grosser malformations established preternatural openings for the intestinal contents, such as umbilical anus, or vulval or vesical openings for the faecal discharge. Others again noticed and recorded Nature's surgical methods of cure in strangulated herniæ, when she established by sloughing and inflammation an artificial anal site. The anatomy of such openings was well known, and the afferent and efferent loop and the spur were described by many ancient writers. It is not difficult to believe that now and then some bold spirit dreamed of emulating so great a teacher and of establishing an artificial anus in those cases where obstruction was threatening the life of the patient. Indeed, to stab the colon of a sheep or horse suffering from obstruction is a veterinary operation whose origin is lost in remote antiquity.

We read in Coelius Aurelianus that Praxagore, who lived four hundred years before Christ, after uselessly employing various means for the cure of ileus, made an opening in the belly and incised the bowel to evacuate the contents, and then closed the wound so made. Professor Fine, writing from Geneva in 1797, tells us that in "ancient times" it was the custom to open the stomach to remove knives that had been accidentally swallowed. The same writer speaks in praise of incising a strangulated hernia to give issue to the fæces.

The first deliberately recorded suggestion for enterostomy, however, is that of Littre reported by Fontanelle, the historian to the Académie Royale de Sciences. This erudite compiler, writing in 1710 in the reign of Louis XIV, states as follows (extract reduced) —

M. Littre saw in the dead body of an infant of six days a maldevelopment of the rectum. The rectum was divided into two portions both closed and connected by only a few threads of tissue of about an inch long. The upper portion of the closed bowel was filled with meconium. The lower portion was entirely empty. "M. Littre, wishing to render his observations useful, imagined and proposed a very delicate operation in the case where one would recognize a similar conformation. It would be necessary to make an incision in the belly, open the two ends of the closed bowel, and stitch them together, or at least to bring the upper part of the bowel to the surface of the belly wall, where it would never close, but perform the function of an anus. Upon this slight suggestion a clever surgeon could imagine for himself details which we suppress. It often suffices to know in general that a thing may be possible and not to despair of it at first sight."

This idea, so magnificently dreamed and so simply stated, was doomed to lie fallow for sixty-six years. We then read how Pillore, a surgeon at Rouen, performed

cæcostomy for a cancer of the rectum The account which here follows was found in the papers of M Pillore, sen, by his son, who looked for them upon the earnest inquiry of M Amussat, who knew by hearsay of the operation Considering the time of its compilation and the place of his practice—remote from seats of learning—it is a masterpiece of clinical writing and surgical judgement and daring in the practice of a country surgeon What follows is a literal translation of the elder Pillore's memoir Let us here rescue his name and deed from surgical oblivion

M Morel, a wine merchant and posting master of Vert-Gallant in the district of Brail, was in the course of the year 1776 taken with difficulty in going to stool He at first experienced some slight pain in the anal region These pains became a little greater, without, however, becoming insupportable, but the difficulty with his motions increased to such a degree that he became anxious and determined to come to Rouen for consultation and the necessary remedies He presented himself to M Delaroche, a capable physician, who ordered him laxatives and gentle purgatives These softened the bowel contents and relieved him for some time But finally, as his difficulties increased daily, he was advised to make use of mercury (or quicksilver) in sufficiently large doses, that by their mass they would overcome the obstacle in the bowel The patient indeed took two pounds of quicksilver It was watched for every day but did not appear The motions became totally suppressed and the belly increased in size from day to day, without, however being tender or inflamed In this state of affairs I was consulted (It was now a month since the patient had taken the mercury without having passed a single drop of it)

I first examined the rectum, thinking indeed it was there the obstruction would be found believing it was possibly formed by hardened and incarcerated fæces as I had often seen to happen, but instead of this species of obstruction I found the upper part of the bowel fixed and scirrhus, forming a very large tumour which totally obstructed the rectum I tried to pass sounds and cannulæ of all shapes and sizes, continuing my efforts for several days, but uselessly In this state—that is to say, the patient having passed nothing from the bowel for over a month, and his belly enlarging daily in spite of his most austere diet—I proposed to him that I should make him an artificial anus He agreed with me and cited the case of a man in his village who for several years had had an artificial anus which Nature had provided following a strangulated hernia I knew of this case, also of another in a woman and from the same cause

I was then indeed determined to perform the operation, but as the case was a very delicate one I first asked five or six of my colleagues to see the patient in consultation with me No one was of my opinion and no one agreed with me But the patient, a man of great sense, being present at our consultation, prayed my colleagues to show him any other means by which he might be saved They answered that they knew of none "Very well," he replied, "it is indeed imperative to operate since my illness is mortal and you know of no other means to save me"

Encouraged by so strong an argument I performed the operation in the presence of my confreres, and of six pensioned pupils who were with me at the time I chose the cæcum as that part of the bowel most suited to our need, as much by its situation as because it would furnish a reservoir, and by its continual and involuntary action would hasten the evacuation of the intestinal contents A small plate furnished with a sponge in the shape of a large button and held by an elastic bandage was devised in place of a sphincter, so that the patient could at all times voluntarily remove it when he felt the need, and, by means of a small clyster, he could from time to time cleanse out the reservoir My patient and I conferred together and thought of all these things before the operation I then operated

I commenced with a transverse incision a little above the groin which I deepened above and below to the depth of the cellular tissue I arrived at the aponeurosis of the external oblique which I incised to the same extent a little above the Fallopiian ligament (Poupart's) in order to have at least a good inch of space from the integuments to the cæcum I made a transverse opening in the muscles and peritoneum almost to the same extent The base of the cæcum, easy to recognize by its appendix, presented itself—I did not have to search for it I drew the cæcum out as far as possible and without effort, there held by an assistant and myself, I opened it transversely and stitched it to the two lips of the wound by means of a thread on two needles which I passed from one side to the other I passed them from

within outwards and pulled out the thread in the middle, thus obtaining two ligatures which I tied above and below over two compresses to press together the edges of the wound. The contents of the bowel came out in abundance. For a dressing I applied burnt charcoal and towels. I used no pressure in order that the issue of faecal matter might not be interrupted. In fact they ran out in abundance for several days, and the belly diminished considerably in size. As the quicksilver was giving us anxiety and we had not seen a single drop of it appear, we caused the patient to be put in all possible positions that might give it an easy issue. There was not the slightest sign of it, however. Fourteen or fifteen days had passed since the operation, during which time the wound had supplicated and the bowel was glued to the skin. I had taken out the sutures and all appeared to be in the best possible state when the patient reported vague pains in different parts of the belly. We at first attributed it to gases shut in the intestines, but the patient, uneasy, said always that the pains were due to the mercury and consequently continued to take such positions as might help it to come out. On the twentieth day the belly, which had been very flat, became swollen and painful. Emollient fomentations were applied, and through our artificial anus we threw some injections into the colon. He was bled twice, but in spite of all our efforts the symptoms quickly augmented and the patient died on the twenty-eighth day after his operation.

I performed the autopsy in the presence of the same surgeons, colleagues, and pupils, and found as follows —

The cæcum and the whole of the colon were healthy and in good condition. The cæcum was adherent to the lips of the wounds, except in one angle where there was a small area of suppuration in the neighbouring cellular tissue, which did not, however, communicate within. The colon was open to the whole of its extent, and only contained some glairy mucus. The cancerous obstruction which was the primary illness was eight or nine inches long, situated at the end of the colon and the beginning of the rectum, totally obliterating the intestinal canal. The tissues surrounding the rectum were hard and fixed. At the side of the rectum was an opening whose calloused edges announced it to be a species of chancre from which issued faecal and purulent material. The peritoneum in the neighbourhood of the kidney was inflamed, without, however, having supplicated. The peritoneum was inflamed and adherent to the folds of the intestines. The quicksilver which the patient had taken was found in one of the last convolutions of the jejunum, which it had dragged down by its weight to the pelvis, behind the bladder. It was pocketed in that portion of the bowel which contained it. This bowel presented here and there gangrenous areas, and was inflamed, the inflammation extending to the loins. The mercury was all recovered and it had not lost a bit of its weight. We believe that we could conclude that if the operation has not met our expectations for its success, it was because of the mercury. For it is very probable that when the intestines, which because of their great dilatation had lost their power of action, became empty of stercoral material, their peristaltic action was not sufficiently powerful to move the mercury. Then followed inverted and retrograde movements, as announced by the nausea and colic which the patient experienced on the twentieth day of his illness. Considering the pull on the mesentery and intestines by this mass of two pounds, one is not surprised that gangrenous inflammation occurred and produced the death of the patient.

Surely a remarkable case and one which any modern surgeon would not be ashamed to report.

Allan, the recorder to the *Recueil Periodique de la Societe de Medecine de Paris*, writing in 1783, tells us that by *hearsay* he learnt that Dubois performed Littre's operation for an imperforate anus in a child of three days. The child died on the tenth day.

Colostomy, however, had its real birth when Duret in 1793 performed successfully left iliac colostomy for a case of imperforate anus in a child three days old. Here is his account —

Friday, Oct. 18, 1793, Marie Poulouen, midwife of Brest, delivered the wife of Michel Ledreves, labourer, of a child. She noticed the infant had no anus and that the sexual parts were malformed, judging that in this state of affairs the child had not long to live she advised the parents to bring the child to Brest to receive surgical aid. On Saturday at

ten in the morning the father came to my house and I examined the child. The sexual organs were so formed that the scrotum was divided at the raphe into two equal parts, each containing a testicle. At first sight one believed the child to be a female. The glans penis lay upon the perineum pierced by the urinary meatus from which urine issued freely. The region of the anus showed no sign of the existence of a rectum. The skin was of natural consistency and colour, and no tumour presented when the child strained. After making this examination I believed the case to merit the attention of those most skilled in the art of healing, and with this in view I called a consultation of all the physicians and surgeons attached to the various hospitals of the city. The consultants advised opening the skin at the spot where the rectum should be present and searching for the bowel. The operation was not successful. I was able to appreciate by passing a sound through the wound into the pelvis that the lower portion of the big bowel was absolutely missing. It was now four in the afternoon and the infant appeared without resource. The vomiting, the extraordinary swelling of the belly, the coldness of the lower limbs seemed signs of certain death. To my surprise, however, the next morning the child still lived. This decided me to call a second consultation, at which I proposed as a last resort, to prolong the life of the child the performance of laparotomy and the establishing of an artificial anus. To give me confidence in this most extraordinary procedure I performed it upon the dead body of a child of fifteen days which I took from the poorhouse of the city. I made an incision on the left side between the last of the false ribs and the iliac crest about two inches long. I exposed the pole of the kidney and a portion of the left side of the colon, this last was opened. I then injected some water by the anus. A portion of the fluid came out through the opening of the colon and a portion escaped into the belly. I then recognized by opening the belly that in the fœtus the lateral areas of the colon are not extraperitoneal as in the adult, but that the colon has a mesocolon which renders it free and floating. This circumstance caused me to reject the operation in this region in the fear that it would give rise to an escape of meconium into the belly. Those assembled after witnessing this trial and after prolonging the discussion sufficiently to prove both its interest to humanity and to surgery, decided (1) That without some extraordinary intervention the death of the child was inevitable, (2) That the axiom of Celsus, "that it is better to employ a doubtful remedy than to condemn the patient in certain death", here found its application, and finally (3) That the decisions of M. Hevin upon laparotomy were not transgressed by this operation, as the cause and course of the malady were, as here, recognized.

I opened the belly of the little patient in the left iliac region in the neighbourhood where the sigmoid colon was forming a tumour a little apparent to the eye and where the meconium already imparted a slightly deeper colour to the skin. I made an opening about an inch and a half long which served for me to introduce the index finger into the belly, with which I lifted and pulled out the sigmoid colon. In the fear that it would immediately fall back into the belly I stitched it by two wax threads passed through the mesocolon. I then opened the colon longitudinally. Gas and meconium came out in abundance. When the bowel had emptied itself to a certain extent I applied a dressing. It was simple and was composed of a pierced compress. In the night between Sunday and Monday the baby slept well, the body heat returned, the vomiting ceased, and the child took the breast easily on several occasions. The day following the operation all who had witnessed the operation the evening before expressed themselves satisfied with the advantageous changes they perceived. The bandages which surrounded the child were filled with meconium, and his voice, which had previously hardly been distinguishable, was now heard lustily.

On the third day, as things were going from better to better, I charged the parents to bring the child twice daily to the hospital. Citizen Massac, Chief of the Administration, and Citizen Coulon, Physician in Chief, were charged to provide the necessary dressings.

On the fourth day the stools became yellow and less in quantity, so I ordered a washing out with simple water and two drops of syrup of rhubarb. This produced a good effect and gave the patient several stools.

On the fifth day the threads which held the bowel appeared useless, so I removed them, for they were already producing redness and irritation in the region of the artificial anus.

On the sixth day about an inch of the internal coats of the bowel appeared through the opening, giving the wound the appearance of a chicken's egg. I attempted to reduce the prolapse by passing a lead cannula into the fistula, both to obstruct any further herniation and keep a free passage for the feces, but the child's cries made one defer this means. The

instrument has, however, since then been perfected by Citizen Morier, a clever cutler of this city. On the seventh day the child was so well, both at the site of operation and in exercise of his functions, that I judged him no longer in need of care or supervision by a person of art.

It is interesting to note that this little patient lived to the age of forty-five.

With Duret begins the history of colostomy. Observe (1) Duret antedated Callisen in the conception of lumbar colostomy, (2) Duret was ignorant of Littre's academic suggestion of left iliac colostomy, (3) He made a very small incision, (4) He made the bowel secure by a stitch in the mesocolon, thus obviating recession of the bowel—in this idea he anticipated the proceeding of Allingham, jun., by over ninety years, (5) He noted the occurrence of prolapse, (6) He used the artificial anus as a channel for the administration of a colonic wash-out. Surely, as the prophet says, "All knowledge is but a remembrance, and all discovery but a forgetting."

For all that Duret was a Professor of Surgery at the Military and Marine Hospital at Brest, he was but a humble and obscure naval surgeon practising in a remote seaport, and his experiments, his operation, and his remarks were lost to surgical knowledge until rescued by Amussat some forty years later. He had the bitterness to know that his thunder was stolen by a contemporary, one Dumas, Professor of Medicine at l'Ecole de Sante de Montpellier.

Dumas in 1797 reported to the Medical Society of Paris a case of imperforate anus. The surgeon Estor, he stated, made repeated attempts to find the rectum from the perineum but failed. Dumas then suggested to Estor the practicability of establishing an artificial anus in the left iliac region. Dumas states that Estor replied, "I approve of the idea, but I have not the courage to execute it. The parents are against it and I shall compromise my reputation by practising so severe an operation of which the success seems so uncertain." The child died on the third day and Dumas made an autopsy. He made an incision in the left iliac region, the colon was distended with gas and liquid and occupied a considerable space. It was pressing hard against the belly wall. Dumas states it would have been very easy to make the artificial anus which would have prolonged the patient's life and "which would have been a rare and interesting contribution to science." States Dumas, "Past historians have contented themselves with describing the malformations of the anus and the rectum, but they give no indication for a cure." He pleaded that the method of Nature as seen in preternatural anus and in the artificial anus following strangulated hernia is an indication of the method surgical art should employ. He stated that the "iliac passion" following upon obstruction of the rectum could be relieved by this means. The operation he suggested was an opening of the belly in the region of the lower end of the colon, then to open the intestine and stitch the opening by means of a thread. The incision ought to follow the direction of the muscle fibres, which would preserve a sphincteric action and avoid either collapse or prolapse of the bowel.

Dumas held a high position. He was a professor of medicine in a big school. That he was by no means ignorant of the literature of his time is conclusively proved by the two pages of quotations and references upon rectal malformations which he gives in his essay. It is incredible for us to believe that he had not read of the proposals of Littre, and still more incredible that he was ignorant of the report of Duret's case which was published in full in a medical periodical which was distributed to the whole of Europe. Indeed, were he ignorant of both these

proceedings it is hard to believe that he had not heard of the operation already performed by Desault, Surgeon-in-Chief to l'Hôtel Dieu in Paris, who in 1794 performed Littre's operation in a case of imperforate anus two days old. It is interesting to note that Desault did not suture his wound. He operated through an incision $3\frac{1}{2}$ in in length. After opening the bowel he stuffed in a dressing with the object of "keeping open the passage for the meconium and causing the edges of the wound to stick." The child died in four days. The specimen was modelled in wax and is in Desault's Museum.

No Dumas stole the thunder of Duret and maintained a discreet silence regarding Duret's operation, and appeared before the Medical Society of Paris in borrowed plumes.

This case of Dumas', however, upon which he did not operate, created great attention in France and Europe generally. It was the subject of a review by Allan, the Recorder of the Medical Society of Paris, and by Martin, the Recorder of the Medical Society of Lyons, who were more generous in their appreciations of the surgeons who had actually dared to operate. It was the consensus of medical opinion that the inguinal incision of Littre was infinitely preferable to the vertical lumbar incision of Duret and Callisen. They believed that it was an impossibility to approach the colon posteriorly without opening the peritoneal cavity. Allan alone of all commentators does not despair at this time of the lumbar route, and states in 1797 "that the lumbar route merits consideration, and, if it could be rendered safe and easy, the results would be infinitely less disagreeable than those of the abdominal or iliac route."

The next step forward in the history of colostomy leads us to Geneva. In 1797 Professor Fine, Surgeon-in-Chief to the Hospital of Geneva, performed transverse colostomy in a woman aged 63 years for acute obstruction due to a cancer of the rectum. Fine stated that for many years he had been struck by the insufficiency of medical means in tympanites and had long been determined to perform enterostomy. He had often advised it but had never been allowed to perform it. In the case reported he had intended to open the ileum, as he states it is here that Nature most often makes an artificial anus in strangulated hernia. His patient being fourteen days obstructed, he opened her in the middle line below the umbilicus, drew out a loop of inflamed bowel, passed a stitch through its mesentery, and sewed it to the skin. His operation was successful. The patient died three months later, when he discovered at the autopsy that he had performed a transverse colostomy. The patient had a growth in the upper part of the rectum causing complete obstruction. In his report he stated that he was ignorant of Dumas' paper and only received his copy of the medical periodical two weeks after having performed this operation. Fine's two memoirs are models of correctness. Professor Fine was conversant with all the history of this time and acknowledges all previous operators and writers. He gives generous tribute to Dumas for his idea of enterostomy for cancerous obstruction.

We now leave France in our pursuit of colostomy and turn to Copenhagen. Here we find that Callisen, Professor of Surgery at Copenhagen, in writing upon imperforate anus states, "If the intestines (rectum) cannot be reached either with the knife or a trocar, it will indeed be difficult to save the patient. An incision of the cæcum or the descending colon has been proposed in these cases, by means of an incision in the left lumbar region at the border of the quadratus lumborum

to establish an artificial anus, the chance of success is very slight and the life of the little patient can hardly be saved. However, the intestine ought to be more easily reached in this spot than from the inguinal region." This observation is to be found in Callisen's *Systema Chirurgæ Hodiernæ* (p. 688) published in 1800. Sabatier, writing in 1810, states "Instead of making an opening in the belly above the groin, M. Callisen, a surgeon who enjoys a distinguished reputation at Copenhagen, has proposed to search for the descending colon in the left lumbar region, where he supposes that it is to some degree outside the general peritoneum cavity. Callisen attempted this operation on the dead body of an infant with an imperforate anus. He did not carefully observe his landmarks, with the result that he opened the peritoneal cavity and penetrated the abdomen. Having made a second incision more posteriorly he came upon the colon, as he had hoped. However, he does not disguise the fact that his fingers, which he introduced into the first incision to help him secure the bowel, were very useful in performing the posterior operation. The wound which he made does not appear to him to be of great importance in view of the small number of blood-vessels which he encountered."

We note that Callisen does not claim the idea as being original. Yet it is he who has had the credit of the conception of lumbar colostomy until now. It was, however, Duret who first originated this incision in 1793. Callisen thought very little of the lumbar method of approach, as indeed did all who followed him until the time of Amussat in 1839.

The first colostomy in England was practised in 1815 by Freer, a surgeon in Birmingham. Freer was also the first surgeon to tie the external iliac artery successfully. In 1815 he performed left iliac colostomy for imperforate anus. The child lived three weeks, dying eventually of marasmus. In 1817 he performed left iliac colostomy in a man of 45 suffering from complete obstruction due to what appears to have been a simple stricture of the rectum. This patient died on the ninth day. His colostomy had acted freely for the whole time. The patient had been purged every day and bled once. From a perusal of the notes of this case he seems to have died of a rupture of the cæcum induced by excessive purgation. This accident is not unknown at the present day. Freer's incision was an oblique one in the left iliac fossa. The colon was stitched to the wound, the stitches were removed on the fifth day, as by then the gut was adherent to the wound. On the eighth day the bowel prolapsed. Several enemata were given through the artificial anus. Freer states that the opening in the belly was made very small to prevent extrusion of the bowels, as this was an accident which had frequently happened to him when operating on dogs.

The next operation in England was performed by Daniel Pring, a surgeon of Bath, who in 1820 performed left iliac colostomy for a patient who was twelve days obstructed from cancer of the rectum. He was led to perform the operation in imitation of the artificial anus which follows the natural cure of strangulated hernia. He was ignorant at the time of any previous writing upon the subject. His patient lived. Pring concludes his memoir as follows: (1) The operation prolongs life in cancer of the rectum, (2) It is always applicable in imperforate anus, (3) It would be useful in simple strictures of the bowel, (4) Peritonitis is a danger—but this would not deter him from operating, (5) It is imperative always to open distended bowel, (6) He advises cæcostomy for obstruction in the transverse colon, (7) The opening can be controlled to act once in twenty-four hours, (8)

Prolapse can be prevented and the opening protected by means of a truss and pad. Pring seems to have been a clever, thoughtful, and resourceful surgeon.

No further operations were performed in England until 1824, when Richard Martland, Physician to Blackburn General Dispensary, operated successfully for a chronic intussusception. His operation was quite successful.

It was not, however, until the time of Amussat that colostomy as an operation was rescued from the realm of the occasional and heroic and advanced to its proper place in surgery. Amussat was a clever and well-known surgeon in Paris. He was called to attend the celebrated Professor Broussais in his last illness. He stated that on seeing him die from stercoral tympanites produced by a rectal carcinoma, he determined that never again would he remain a passive spectator of such another death. The death of Broussais, and the examination of two specimens in the Musée Dupuytren—the one a fungating rectal growth, and the other a scirrhus of the rectum removed post mortem from the body of the tragedian Talma—led him to a careful reconsideration of the whole question of artificial anus. He collected the statistics of all cases from Pillore in 1776 to his own first case in 1839. Over a period of sixty-three years there were 29 cases, with a mortality of 20. Of these 29 cases, 21 had been operations for imperforate anus, of which only 4 survived. It is curious to note that all these 4 survivors had been operated upon in Brest, the seaport town in which Duret first performed the operation. The remaining 8 cases were adults, and of these only 5 had survived. All of these 29 cases had been operated upon by the abdominal route. Amussat attributed the death in all cases to peritonitis.

Amussat, in spite of the failures of Duret and Callisen, and the condemnation of all previous writers and the surgeons of his own time, determined to decide for himself the possibility of opening the colon posteriorly without wounding the peritoneum. By a series of dissections he showed conclusively that Duret and Callisen, while recognizing the peritoneal relationships of the colon in the lumbar region, failed to appreciate the possibilities of lumbar colostomy in the adult. He demonstrated the existence of large cellular spaces in the lumbar regions and in the pelvic mesocolon, and that these spaces are greatly developed both in children and adults under conditions of obstruction, or of deliberate distension by means of a bellows and tube. He showed that both in the infant and adult lumbar colostomy was at all times possible.

Amussat abandoned the longitudinal incision of Duret and Callisen. He operated by a transverse incision 4 in. long midway between the last rib and the iliac crest, and extending well back to the erector spinæ and the quadratus lumborum muscles. The bowel was fixed at the anterior angle of the wound by a stitch and the rest of the wound left open. He operated without assistance, and twisted the arteries instead of tying them, as the long ligatures which were then used got in his way. His guide to the colon was the pad of fat at the outer edge of the quadratus, and his guide to the point to open the bowel was the 'yellow spot' where the peritoneum is reflected to the abdominal parietes. If obstruction was not already present, Amussat distended the bowel by means of a bellows and tube. He contended that the operation was as urgently indicated in cases of obstruction of the big bowel as was herniotomy in strangulation. He determined the site of obstruction by (1) Rectal examination, (2) Estimation of the amount of fluid it was possible to inject in the rectum, and (3) In some cases

when the exact site of the tumour could not be determined he punctured the distended bowel with a small trocar

Amussat advised operation on the right side (1) When the tumour was too near the site of operation on the left side, (2) When the obstruction was far from the anus, and (3) When the site of obstruction could not be determined

He states that in right-sided lumbar colostomy the retrograde peristalsis of the colon distal to the opening is a source of distress, and that "it would be advantageous to form some sort of septum to prevent the fæces passing beyond the opening" This, however, he could not do, as he states the intestine is so strongly fixed to the edges of the wound that to form a spur would be impracticable and dangerous He states also that there is no tendency to prolapse in his method, and that the smallness of the opening, which has always a tendency to close and retract, prevents the continuous and involuntary escape of fæces He performed the operation nine times successfully

Lumbar colostomy was an operation well suited to the pre-anæsthetic and pre-antiseptic times in which Amussat lived His activities gave the operation an impetus which carried it successfully to the time of the younger Allingham It became until Allingham's time the operation of election, and was practised by all surgeons in Europe, England, and America

No proctologist should be in ignorance of Amussat's writings His memoirs deserve to be read by all surgeons They are models of all that surgical communications should be in honesty, completeness, conciseness, and well-reasoned judgement

Amussat concludes "An artificial anus, it is true, is a grave infirmity, but it is not insupportable To be able to practise it a surgeon ought to fear to be surprised by a pressing occasion, and he should prepare himself by many repetitions of the operation upon the cadaver"

The first operation of lumbar colostomy in England was performed by Clement, of Shrewsbury, who in 1841 did right lumbar colostomy for a whipcord stricture of the transverse colon The patient lived three years Lumbar colostomy, however, owes its real introduction in England to John Erichsen, Surgeon to University College Hospital Erichsen was a pupil of Amussat and witnessed Amussat's first operation Hence his prejudice in its favour In a report published in 1841 he gave Amussat's writings to the English medical public He advised the operation for (1) Imperforate anus, (2) Simple retention of fæces which could not otherwise be relieved, (3) Obstruction of the big bowel, and (4) In cancer of the rectum as soon as pain becomes severe He claims for the operation that the wound is easy to dress, that its posterior situation is less distressing than is the inguinal, and that the absence of the spur is indeed an advantage, as the wound may close naturally

A further extension of the applicability of lumbar colostomy is seen when Pennell in 1849 performed it for rectovesical fistula, following a rectal stricture and a fistula in ano His patient lived for fifteen years Croker Pennell was a Lecturer in Surgery at Westminster Hospital and later was attached to the Faculty of Medicine in Rio de Janeiro In 1850 Avery as Surgeon of Charing Cross Hospital performed left lumbar colostomy for a case of intestinal obstruction of nine days' duration The patient died the next day, and the autopsy showed a volvulus of the cæcum and descending colon This shows the limitation of the operation apart from certain diagnosis as to nature and seat of obstruction Luke,

a Surgeon to the London Hospital, appreciated this fact, and writing in 1850 he stated that lumbar operation was only useful when the seat of the obstruction was definitely recognized. Luke was the first surgeon to perform a pararectus incision and to bring out the bowel in the neighbourhood of the rectus muscle. His colleague, John Adams, of the London Hospital, also operated by a hypogastric incision and brought the bowel through the linea alba. Adams advised operation in all cases of cancer of the rectum before obstruction.

Curling, Consulting Surgeon to the London Hospital and President of the Royal College of Surgeons, expressed himself in 1851 as a strong partisan of the abdominal operation. He advised colostomy for stricture of the rectum and for cancer of the rectum before obstruction. He stated that excision of the rectum for cancer had no place in surgery. He regarded both the lumbar and abdominal operations as equally dangerous, but preferred the abdominal route as being easier, as giving a spur, easier to dress and control, and states that by the abdominal route the bowel can always be found. In 1865, however, he appears to have recanted and he became an ardent advocate of the lumbar operation. He apologetically states that with the lumbar operation there may be an occasional sphincteric action by the lumbar muscles. This communication of Curling's in 1865 gave a tremendous impetus to lumbar colostomy in England, as did also the writings of Cæsar Hawkins in 1852.

The next advance in colostomy was when Nathaniel Ward in 1865 performed lumbar colostomy for rectal cancer as a routine upon recognition of its presence.

There were two staunch advocates in England of lumbar colostomy in the persons of Allingham, sen., and of Thomas Bryant. William Allingham, writing in *St Thomas's Hospital Reports* in 1870, and in his book, *Diseases of the Rectum*, in 1873, advocated lumbar colostomy for insurmountable obstruction or intense pain due to a cancerous growth, and for rectovesical fistula in the male. In all his writings he showed great ignorance of the history of the operation, and denied indeed that Amussat ever performed it.

Thomas Bryant, Surgeon to Guy's Hospital (and a great advocate of the galvano-cautery knife), writing in 1874, stated of lumbar colostomy that it prolonged life, eased pain, and prevented aggravation of the disease in cancer of the rectum. He also advised it as the best possible means of treatment in incurable fistula and abscesses of the rectum.

Bryant to the end of his days remained a loyal exponent of the lumbar operation. Speaking in 1889 in his Bradshaw Lecture, delivered at a time when the writings of Allingham, jun., Reeves, and Cripps had revived for all time the operation of inguinal colostomy, he stated that the abdominal operation was not proved superior to the lumbar operation. His lecture, indeed, is a thinly veiled attack delivered by an old conservative surgeon against younger and more active men who were carrying the banner of surgery one step forward. Bryant operated by an oblique incision. He claimed for his operation that it was safe and gave a good spur. He operated to relieve symptoms and did not wait for obstruction to appear.

The 1880's witnessed an increasing interest in the abdominal operation. Peritonitis was ceasing to be the surgeon's bane. Abdominal surgery was making rapid progress, and it is but natural that surgeons gave an added attention to the possibilities of colostomy by the abdominal route. The operation was begging to be better understood, and many surgeons have given their names to proceedings

which testify to their ingenuity in devising methods of spur formation, the prevention of prolapse, and the establishment, with varying success, of some form of sphincteric control. It will be remembered that Duret stitched the mesocolon. This most effectually prevented collapse of the bowel and formed a good spur. Although in 1833 Dupuytren, reviewing the work of Petit, Scarpa, and others, established for all time the characteristics of an artificial anus with its afferent and efferent loops and its spur, and distinguished artificial anus from fæcal fistula, it was not until the time of Allingham and Bryant that a definite effort was made to establish any sort of spur, and this only in lumbar colostomy, and with indifferent success. Jones in 1866 suggested formation of a spur in lumbar colostomy by dissecting up the mucous membrane and stitching it to form a bridge between the two loops. Later on Madelung suggested complete division of the bowel with closure of the lower loop, which he allowed to fall back into the wound. In 1841 Schtizinger applied the principle of closure and invagination of the lower loop to the abdominal colostomies. It was not until 1855 that this primal necessity of a spur was definitely recognized, when Knie, a surgeon in Moscow, experimenting on dogs, found that he could make a slit in the mesocolon, through which he was able to join all the layers of the belly wall. This idea he used successfully in operation upon the human being. In 1887 Ball, of Dublin, in describing his operation for colostomy stated that he always stitched the mesocolon to the skin. This, Ball states, he did to prevent recession of the bowel, not, however, with the view to the formation of a spur. In 1887 Allingham, jun., definitely described the mesocolic stitch. His stitch was a half mattress securing the mesocolon to the skin. He describes it as an essential part of his operation. It is curious that Cripps, writing at the same time, did not appreciate or use this mesocolic stitch. In 1888 Maydl advanced and simplified colostomy with his idea of passing a vulcanite rod through the mesocolon. This rod lay upon the belly wall and very effectually prevented recession of the bowel. Kelsey, a well-known New York surgeon, in 1889 described independently a method of securing a spur and preventing recession of the bowel. He passed a hare-lip pin through all layers of the abdominal wall on one side, then through the mesocolon, and then through the abdominal wall on the other side. Reeves, a Surgeon to the London Hospital, described a somewhat similar method in 1892. Paul, writing in 1890, said that he considered lumbar colostomy "prompt, safe and suitable" to relieve acute obstruction. He considered the inguinal route easier, but, in acute cases, believed it to be a great source of danger because of the unavoidable risk of fæcal infection. He then described his tube, which he tied in the proximal end of the bowel.

In Littre's original suggestion in 1710 he advocated in imperforate anus that the closed proximal end of the bowel should be brought to the skin, "to make for ever" an artificial anus. In the endeavour to perfect colostomy, to obviate the spur, and prevent prolapse, surgeons began to practise complete division of the bowel with closure of the distal end, which was allowed to fall back into the abdomen, whilst the proximal end was secured to the belly wall. This proceeding, first practised by Schtizinger in 1881, then by Senn, of America, was in 1889 adopted by Jessop and Purcell, by Paul, Braun, and Weir in 1890, and with various modifications by Ryall and Lilienthal, and in 1909 by Marro, of Turin.

Allingham, jun., Cripps, and Reeves re-established inguinal colotomy, not forgetting the writings of Ball, of Dublin, and of Davies-Colley, Surgeon to Guy's

Hospital Davies-Colley made two valuable and original suggestions. The first was that of delayed opening of the bowel—the operation ‘a deux temps’. The second suggestion he made was that of exteriorization of the colon when it is the seat of a malignant growth. Davies-Colley deserves well to be remembered for so ingenious a suggestion. It was enthusiastically adopted by Verneul, of Paris, and is described in full in the writing of Reclus. Verneul, by the way, was accustomed to stitch together for the extent of some inches both loops of the bowel, so that on being opened they presented the appearance of a double-barrelled gun. Ball, of Dublin, writes in 1887 as an advocate of what he terms ‘laparo-colostomy’. He believed in an abdominal exploration before colostomy. He stated that it was easier to find the bowel, that the exact spot for the colostomy could be selected, and that the operation was made easier by exploration.

We now recognize that there have evolved two distinct methods of performing an abdominal colostomy. The one depends for its efficiency upon a spur—the method of Duret, Allingham, Knie, Madyl, Kelsey, and Reeves. The other method is that of division of the bowel, closure, and invagination of the distal end, and utilization, in various ways, of the freed proximal loop. It was, by the way, the practice of this latter method which led surgeons to a careful investigation and appreciation of the colonic blood-supply. Many ingenious operations have been devised to obtain sphincteric control of the anus which result from this method. Meanwhile in our consideration of the first category we must not forget to mention the names of Ward, of Leeds, Hartmann, of Paris, and Miles, of London. Ward, who was a colleague of Lord Moynihan of Leeds, devised a suture known as Ward’s mattress suture. It is a further step in Allingham’s mesocolic suture. Hartmann operates through a muscle-splitting incision such as is employed in McBurney’s operation. He forms his spur by passing a roll of gauze through the mesocolon, after the method of Zuckerkandl. And latest of all is the method of Miles, who, first of all, explores the abdomen through a median subumbilical incision and mobilizes the proximal portion of the pelvic colon by dividing any adhesions that may exist. In this way he ensures that the segment of pelvic colon utilized for the colostomy adjoins the descending colon. The loop of colon so mobilized is withdrawn from the abdomen through a small stab incision situated at the junction of the outer and middle thirds of a line drawn from the umbilicus to the left anterior superior spine of the ilium. A loop of stout silk is passed round the mesenteric vessels and then through a loop of silkworm gut inserted through the skin at a point midway between the umbilicus and the stab incision and tied, not so tightly as to occlude the arteries but tightly enough partially to obstruct the venous return. In this way a solid œdema of the spur is produced which effectively prevents it from receding.

BIBLIOGRAPHY

- ADAMS, J, *Med-Chir Trans*, 1851, *xxxv*, 62
 ALLAN, *Recueil periodique de la Soc de Med de Paris*, 1797-8, *iii*, 123
 ALLINGHAM, Jun, *Brit Med Jour*, 1887, *ii*, 874, *Colotomy*, 1892
 ALLINGHAM, W, *St Thomas’s Hosp Rep*, 1870, *i*, *ns*, 285, *Diseases of the Rectum*, 1873
 AMUSSAT, J Z, *Acad Roy de Med*, 1839, Oct, *Ibid*, 1841, Sept, *Ibid*, 1842, July, *Med Times*, 1844, *v*, 401, 425, 441
 AVERY, *Lancet*, 1850, *i*, 607

- BALL, C B, *Trans Acad Med Ireland*, 1887, v, 178
 BATT, W R, *Amer Jour Med Sci*, 1884, 423
 BRAQUEHAYE, J, *Jour de Med de Bordeaux*, 1889-90, xix, 304
 BRAUN, *Deut Gesellschaft f Chir*, 1891, xx, 368
 BRYANT, T, *Lancet*, 1868, 1, 193, *Ibid*, 1874, 1, 52, *Ibid*, 1875, 11, 418, *The Bradshaw Lecture*, 1889
 CALLISEN (Reported by Sabatier), *Systema Chirurgiæ Hodiernæ*, 1800, 688
 CLEMENT, W, *Med-Chir Trans*, 1852, xxxv, 209
 CRIPPS, H, *Brit Med Jour*, 1888, 11, 760
 CURLING, T B, *Diseases of the Rectum*, 1851 and 1876, London, *Trans Roy Med and Chir Soc*, 1860, xliii, *Lancet*, 1865, 1, 3
 DAVIES-COLLEY, *Trans Clin Soc*, 1885, xviii, 204
 DEAYER, *Proc Phil Co Med Soc*, Philadelphia, 1891, xii, 97
 DESAULT, P J, *Journal de Chirurgie de Desault*, 1794, iv, 248, *Œuvres chirurgicales* 3rd ed, 1813, 11, 352
 DIDOT, A, *Memoires, etc*, Bruxelles, 1847, 11
 DUBOIS, *Recueil periodique de la Soc de Med de Paris*, 1797-8, 111, 123
 DUMAS, C L, *Ibid*, 47
 DUPUYTREN, *Gaz des Hôp de Paris*, 1833, vii, 169
 DURET, C, *Recueil periodique de la Soc de Med de Paris*, 1798, iv, 45
 ERICHSEN, *London Med Gaz*, 1841, 11, 189, 223
 FINE, *Ann de la Soc de Montpellier*, 1797, vi, 34
 FREER, *Med and Physic Jour of London*, 1821-2, xlv, 11
 FRIEDBERG, *Arch gen de Med de Paris*, 1857, 1, 565, *Ibid*, 11, 42
 HARTMANN, H, *Rev de Chir*, 1900, Nov, 613
 HAWKINS, CÆSAR, *Med-Chir Trans*, 1852, xxxv, 85
 HEATH, C, *Brit Med Jour*, 1877, 11, 751, *Lancet*, 1892, 1, 1178
 HOWSE, *Holme's System of Surgery*, 1, 801
 JESSETT, F B, *Brit Med Jour*, 1889, 11, 1306
 JESSOP, T R, *Ibid*, 1879, 11, 614
 JONES, P, *Ibid*, 1886, 1, 782
 JONES, R, *Ibid*, 1892, 1, 117
 KELSEY, *Med Record*, 1889, xxxvi, 398
 KNIE, *Zentralb f Chir*, 1885, May 5
 DE LATOUR, H A, *Trans Pathol Soc Lond*, 1879-80, xvi, 386
 LILIENTHAL, H, *Ann of Surg*, 1910, 111, 384
 LITTRE, *L'Histoire de l'Academie de Science*, 1710, 37
 LUKE, *Med-Chir Trans*, 1850, xxxiv, 263
 MARRO, A, *Ann of Surg*, 1911, 111, 250
 MARTIN, JUN, *Recueil periodique de la Soc de Med de Lyon*, 1798, 189
 MARTLAND, R, *Edin Med and Surg Jour*, 1825, xiv, 271
 MASON, E, *Amer Jour Med Sci*, 1873, 354
 MAYDL, *Zentralb f Chir*, 1888, xlv
 MAYO ROBSON, *Brit Med Jour*, 1892, 1, 65
 MAZERY, L E, *De l'Anus artificiel chez l'Adulte*, Paris, 1870
 MONTGOMERY, *Trans Med Soc Philadelphia*, 1892, xxiii, 135
 PAUL, F T, *Brit Med Jour*, 1891, 11, 118
 PENNELL, C, *Lancet*, 1850, 1, 628
 PETIT, *Union medical*, 1886, 577
 PILLORE, H, *Experience*, 1840, 73, Paris, *Gaz des Hôp de Paris*, 1840, 2^{me} S, 22
 PRYAGORE (400 B C), Quoted by Cælius Aurelianus, Lib III, Caput XVII
 PRING, D, *Med and Physic Jour of London*, 1821-2, xlv, 1, *Ibid*, xlvii, 109
 PULOSSON, *Lyon med*, 1884, xlv, 67
 PURCELL, *Illust Med News*, London, 1889, 1, 271
 RECLUS, *Rec de Chir*, 1885, v, 394, *Rec Clin Chir de l'Hôtel Dieu*, 1888
 REEVES, H A, *Brit Med Jour*, 1892, 1, 66
 RYALL, C, *Lancet*, 1909, 11, 15
 SABATIER, *Medecine operatoire*, 2nd ed, 1810, 111, 336, iv, 429, 688
 SCARPA, *Sull' erme (Mem Anat Chir)*, 1809 Milano
 SCHEDE, *Deut med Woch*, Bd xiii
 SIMPSON, *Brit Med Jour*, 1885, 1, 1039
 WARD, *Lancet*, 1865, 1, 3
 WEIR, *New York Med Jour*, 1891, liv, 640, *New York Med Rec*, 1900, 666
 WITZEL, *Zentralb f Chir*, 1894, xl

EXPERIMENTAL SURGERY

THE RELATION OF THE EXTRINSIC NERVES TO THE FUNCTIONAL ACTIVITY OF THE ŒSOPHAGUS*

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THE purpose of this investigation was to determine the exact distribution and function of the extrinsic nerves to the Œsophagus and cardiac sphincter, and thus to obtain if possible a rational basis for the surgical treatment of achalasia

Action of the Vagus on the Œsophagus.—It is generally agreed that vagal stimulation causes an increased 'tonus' of the thoracic Œsophagus, but different workers have obtained varying effects on the cardiac sphincter after vagal stimulation. Openchowski¹ described a 'dilator nerve' to the sphincter. May² and Langley³ showed that the dilator effect was obtainable by stimulation of the vagus as a whole and was not due to a separate dilator nerve. Koennecke⁴ and Rieder⁵ obtained the reverse effect. Veach⁶ reported that sphincter closure followed low-frequency stimulation, while high-frequency stimulation inhibited the sphincter. Carlson, Boyd, and Pearcey⁷ related the variable effects to the coexisting gastric tonus, a point of view which is shared by McCrea, McSwiney, and Stopford.⁸

Reid⁹ in 1839 noted that in rabbits vagal section in the neck caused stasis of the food in the Œsophagus. It is generally agreed that this stasis is due to increased tonus of the cardiac sphincter followed later by dilatation of the Œsophagus (Bernard,¹⁰ Schiff,¹¹ Kronecker and Meltzer,¹² D'Almeida,¹³ and Klee¹⁴). Krehl¹⁵ has described a patulous cardia following vagal section in the neck. McCrea, McSwiney, and Stopford¹⁶ considered that the increased sphincter tonus was only temporary, but it must be noted that in all their experiments the vagi were divided after they had passed into the abdomen.

Carlson,¹⁷ quoting Cannon,¹⁸ stated that after bilateral vagotomy in cats Cannon noted obstruction in some cases for several days and in others indefinitely. Cannon¹⁸ distinguished two different effects of vagal section: (1) paralysis of the upper zone which contains striped muscle, and (2) in the lower unstriped zone paralysis followed by recovery of peristalsis in response to the local stimulus of mechanical distension. In relation to obstruction he stated: "In my experience a difficulty in forcing food through the cardiac sphincter explains to some extent the slower emptying of the gullet during the first few days after operation, which is proved by the fact that strong peristaltic waves, so strong as to produce a very marked bulging of the tube in front of them as they advance, have failed to force food into the stomach. In my experience this usually does not persist as a considerable

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obstacle and the forcing of food into the stomach by œsophageal peristalsis becomes in time not difficult. But there have been instances under my observation in which there was continued trouble. The œsophagus in these cases suffered a marked dilatation and became filled with food which decomposed." Such a description could be interpreted not as that of a transient obstruction but rather as an obstruction overcome by the recovery of peristalsis.

Jurica,¹⁹ in repeating this work, has described the recovery of irregular movements in the upper zone as well, which he believes are mediated through the ganglia of Auerbach's plexus, and which he has demonstrated in this region.

Action of Sympathetic Nerves upon the Œsophagus—Gaskell²⁰ has denied that the œsophagus receives any sympathetic supply, and states that all observers agree that the splanchnic nerves do not affect the œsophagus in causing either contraction or relaxation. Carlson,¹⁷ however, obtained both contraction and relaxation of the cardia on splanchnic stimulation, and Veach²¹ obtained an occasional motor response. Page-May,²² however, could obtain no response.

Kure²³ and his fellow investigators, experimenting on dogs, find that stimulation of the stellate ganglion does not cause contraction of the œsophagus, but that stimulation of this ganglion during vagal stimulation results in an increase of the contraction of the œsophageal wall. Rieder⁵ was unable to obtain this result or any sympathetic response from the œsophagus or cardia. His experiments were conducted in rabbits and dogs.

As regards excision of the sympathetic supply, Jurica¹⁹ found no change in the X-ray appearances of the œsophagus following double splanchnectomy. Kure²⁴ has stated that excision of the stellate ganglia is followed by stoppage of a barium meal in the part of the œsophagus behind the heart, and excision of the sympathetic fibres passing to the cardiac sphincter in an animal in which both vagi had been divided increased the obstruction at this site resulting from the vagal section. On these grounds he postulated synergism of the vagus and sympathetic in the œsophagus. Jurica¹⁹ found that previous double splanchnectomy, followed by bilateral vagal section, does not alter the appearances resulting from the vagal section.

Kuntz²⁵ states that in man the œsophagus is innervated chiefly from the inferior cervical ganglia, further branches also passing to the œsophagus directly from the thoracic sympathetic ganglia, or from these ganglia via the aortic plexus or a plexus in the posterior mediastinum. It is in relation to these direct branches that one discrepancy arises, some authors figuring many and others few or none. Hovelacque²⁶ considers that they are few in number and not to be found in all subjects. Where they occur they are more numerous above the origin of the great splanchnics. With the latter point Testut²⁷ is in agreement.

In regard to the possibility of a recurrent supply from the abdomen, it might well be that those sympathetic fibres which supply the cardia also innervate the adjacent sphincter, and here Brandt²⁸ has described a large branch arising from the semilunar ganglion of the left side passing directly to the cardia. Hovelacque,²⁶ in describing the coronary plexus of the stomach, states that it is distributed to the cardia with the cardio-œsophageal branch of the left gastric artery. Hurst²⁹ observes that branches derived from the splanchnic accompany the inferior phrenic arteries and piercing the diaphragm supply the lower part of the œsophagus.

Nature of the Cardiac Sphincter—This is the subject of controversy. Evidence for the existence of such a structure has been brought forward by Langley,³

and more recently by Poulton and Payne,³⁰ who have observed the contraction occurring at this region in rabbits and have caused relaxation of the contracted region on vagal stimulation. McSwiney³¹ regarded this structure as part of the stomach rather than the œsophagus, on account of the response of its musculature to drugs, the responses resembling gastric rather than œsophageal reaction. Views held by other authors suggest that sphincteric action in this region depends upon the contraction of adjacent structures—e.g., the cruciate fibres of the diaphragm and the so-called liver tunnel, either alone or together with an intrinsic sphincter mechanism. Thus Jackson³² stated “The diaphragmatic pinchcock is the normal mechanism by which, along with kinking of the œsophagus, the food in the stomach is prevented from regurgitation, and the diaphragmatic pinchcock opens at the proper moment in the deglutitory cycle.” Mosher,³³ in describing the action of the surrounding liver in relation to deglutition in the abdominal œsophagus as observed by X rays, writes “In watching a patient with a normal œsophagus swallow, the œsophagus is seen to come to a point momentarily at the upper border of the liver. Then, after a delay of a second or two, the liver tunnel opens up and the milk streams into the stomach.”

Fulde,³⁴ in a recent investigation of the mechanism of the cardia, describes an intrinsic mechanism the action of which is modified by the phrenico-œsophageal membrane. This structure, in virtue of its high attachment to the œsophagus, can close the cardia by compression during descent of the diaphragm, opening therefore occurring at the beginning of expiration. This action is mechanical and depends upon the relative position of cardia and diaphragm.

EXPERIMENTAL METHODS

In attempting to elucidate some of these problems the methods chosen have been anatomical dissections in stillborn children and animal experiments. In animals the methods of investigation were two.

Method 1—Firstly, the study of changes produced in the intra-œsophageal pressure on electrical stimulation of the extrinsic nerves to the œsophagus. The changes of pressure produced were received by a thin rubber bag introduced through the mouth and connected by a tube to a pump by which the bag could be distended, and also to a water manometer recording on a kymograph. Electrical stimulation was carried out using a coil and vulcanite insulated electrodes. The coil was kept at 8 to 10 cm., having four volts in the primary circuit. For sympathetic stimulation a combination of an electric pendulum interrupter in the primary circuit and a metronome in the electrode circuit was arranged so as to produce volleys of six make-and-break shocks occurring three times a second. For vagal stimulation it was found that the results were the same if the vagi were left intact or both divided. The best responses were obtained from the right vagus, and in all experiments vagal stimulation refers to stimulation of the peripheral end of the divided right vagus in the neck, the left remaining intact. Light intratracheal ether anæsthesia was used (with positive pressure when the thorax was opened). In all cases the animals were experimented on twenty hours after their last feed.

Method 2—Secondly, the extrinsic nerves were divided by various operations described below, and later the changes resulting in the œsophagus were studied by means of X-ray plates taken of barium milk introduced by a tube passed from the mouth under ether anæsthesia.

The animals used in all cases were cats. The choice of these animals was made deliberately following the work of Arey and Tremaine³⁵ on the muscle content of the œsophagus in animals and humans. These observers have shown that these animals correspond more closely to the human in that both possess unstriped muscle in the lower third of the œsophagus, whereas dogs differ from the human in containing striped muscle as far as the cardia, and rabbits are unsuitable in possessing three muscular coats. All experimental results have been confirmed in at least three cases.

EXPERIMENTAL RESULTS

METHOD 1

The results obtained from stimulation of the extrinsic nerves were found to vary according to the portion of the œsophagus under examination, and will be described as to vagal and sympathetic stimulation under four zones.

1 Upper Third of Thoracic Œsophagus.—

a Vagal Stimulation—This caused a simple tetanic contraction of the upper third, after a latent period of $\frac{1}{3}$ to $\frac{1}{2}$ second. The contraction passed off immediately stimulation ceased. There were no secondary waves (*Fig 111, v*). The result appeared to be a shortening of the œsophagus and a straightening of its lower end.

b Stimulation of the Stellate Ganglion—If carefully isolated, stimulation of this structure caused no contraction of the œsophagus (*Fig 111, ss*). When, however, this stimulation was combined with stimulation of the vagus there resulted an increase in the degree of contraction of the œsophageal wall (*Fig 111, vs*). Further, if the vagal stimulation was prolonged sufficiently, until the œsophageal pressure showed signs of returning to the normal level, sympathetic stimulation caused an increase in the contraction above the original vagal rise (*Fig 112*).

After prolonged vagal stimulation of approximately 20 seconds followed by an interval of about 15 seconds, sympathetic stimulation alone produced an increase of pressure within the bag (*Fig 112, s*) which showed oscillations exactly similar to the original oscillations occurring at the crest of the combined vagal and sympathetic stimulations.

2 Lower Third of Thoracic Œsophagus.—

a Vagal Stimulation—This was followed by an increase of tonus and motility in the lower third of the œsophagus, persisting for some time after stimulation (*Fig 113*). The latent period of this reaction is approximately $\frac{1}{2}$ second, a second type of response consists of a series of secondary waves, three or four in number, occurring at intervals of approximately 10 seconds and following upon an initial contraction or relaxation.

b Stimulation of the Stellate Ganglion or Thoracic Sympathetic Chains—This caused a slight diminution of tonus and inhibition of the increased motility resulting from vagal stimulation or from mechanical distension of the œsophageal wall by the inflated bag (*Fig 114*).

3 Middle Third of Thoracic Œsophagus—The effects produced were variable and intermediate between the above.

a Vagal Stimulation—This caused tetanic contraction followed by secondary waves, a combination of the effects seen in the upper and lower thirds.

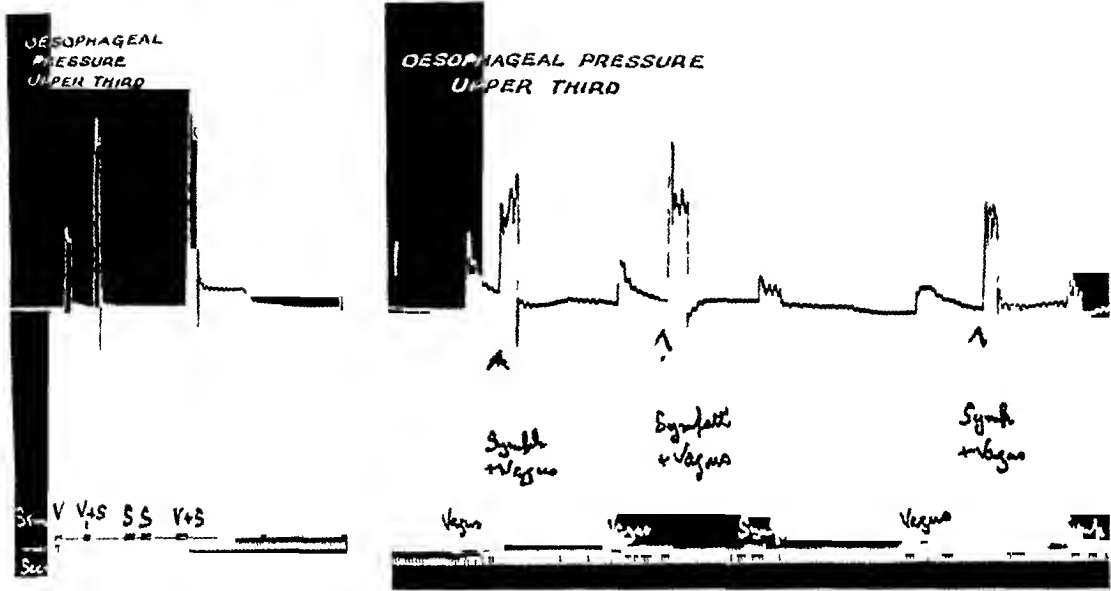
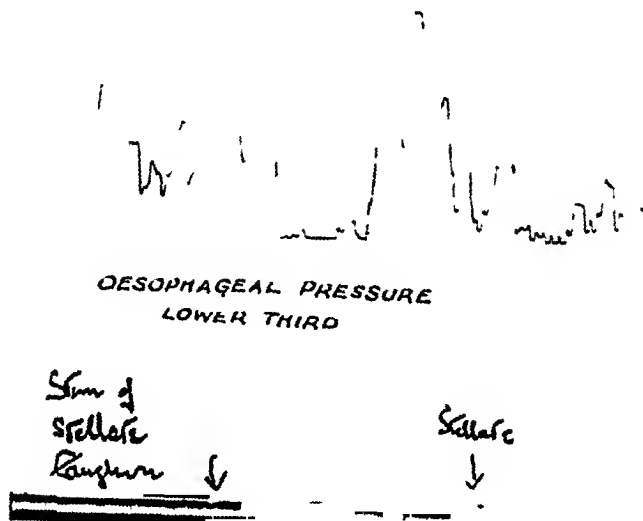


FIG 111 —Tracing of pressure within the upper third of œsophagus, showing the effects of vagal and sympathetic stimulation, separately and combined

FIG 112 —Tracing of pressure within the upper third of œsophagus, showing result of sympathetic stimulation upon prolonged vagal stimulation

1 cm
 5 sec
 STIM. BY VAGUS (P. 1000)

FIG 113 —Tracing of pressure within the lower third of œsophagus, showing prolonged effect of short vagal stimulation



b Sympathetic Stimulation—This caused a diminution of tonus or an increase of the contraction resulting from vagal stimulation. The varying results appeared to be due to the exact position of the intra-oesophageal bag.

4 **Inter-diaphragmatic and Intra-abdominal Portion of Oesophagus**—

a Vagal Stimulation—This caused immediate relaxation of this portion of the oesophagus in all cases (*Fig 115*)

b Stimulation of the Stellate Ganglion, Thoracic Chams, Splanchnic Nerves—No effect (*Fig 115*, STELL)

c Stimulation of the Nerve-fibres surrounding the Cœliac Axis Artery and Left Gastric Artery—A gradually increased contraction of this portion of the oesophagus was observed (*Fig 116*)

Microscopic section of these nerve-trunks showed the presence of medullated fibres among the non-medullated trunks. Reflex effects, such as a complete vomiting reflex and alterations in pulse and blood-pressure, demonstrated the presence of afferent fibres in these trunks. To make sure that the rise of tonus in the oesophageal musculature was not reflex in nature, the experiment was repeated with the artery and nerves divided. Stimulation of the peripheral end still produced a rise in tonus, showing that the effect was not reflex, provided that the strength of the stimulation was increased to compensate for the impaired blood-supply (*Fig 117*)

The above results suggesting the presence of an intrinsic sphincter at this region, the experiments were repeated with the diaphragm divided, the liver displaced, and the oesophagus lying free. Relaxation was still obtained on vagal stimulation and contraction on sympathetic stimulation (*Fig 118*)

METHOD 2

The results of excision of the extrinsic nerves of supply as studied by X-rays of barium introduced into the oesophagus under ether anæsthesia are given below.

The Normal Cat—In studying these plates it is necessary to have for comparison the X-ray appearances of the oesophagus of the normal cat. There is moderate peristalsis of the oesophagus. There is no contraction in the region of the diaphragm and intra-abdominal oesophagus (*Fig 119*). When the meal has entered the stomach it cannot be returned to the oesophagus by digital pressure exerted upon the stomach contents through the abdominal wall (*Fig 120*)

Bilateral Division of Vagi—There is dilatation of the thoracic oesophagus and decreased motility, peristalsis recovering later in the lower third. There is contraction in the region of the diaphragm and abdominal portion—i.e., that region which was shown above to exhibit the reactions of an intrinsic sphincter (*Fig 121*)

If the meal enters the stomach it cannot be returned to the oesophagus by digital pressure (*Fig 122*)

The degree of the changes produced depends upon the level at which the vagi are divided and the time that has elapsed since operation. *Fig 121* shows the condition four months after bilateral division of the vagi below the lung root. Two weeks after operation this animal showed no obstruction at the sphincter. *Fig 131* shows the condition in the same animal seven months after operation, in which the obstruction had increased sufficiently to produce vomiting. If the denervation is more extensive and designed to cut off all vagal fibres, by division of the right vagus in the chest below the recurrent laryngeal, and division of the nerve plexus on the

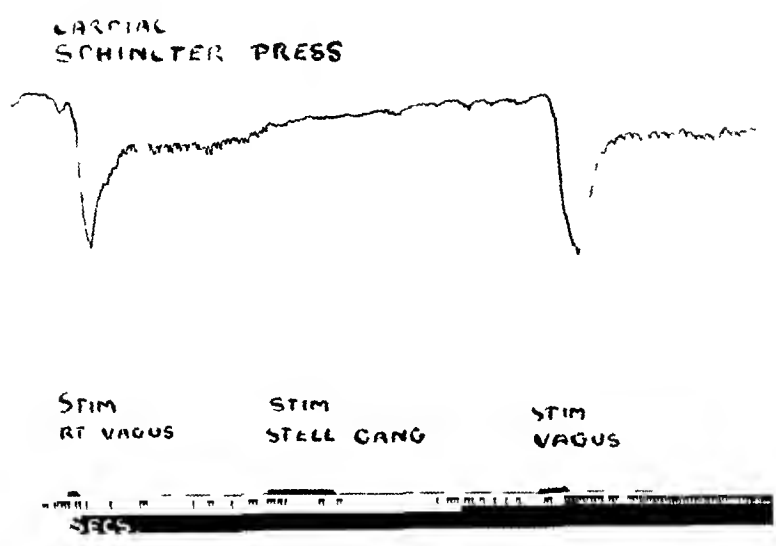


FIG 115—Tracing of cardiac sphincter pressure, showing vagal inhibition

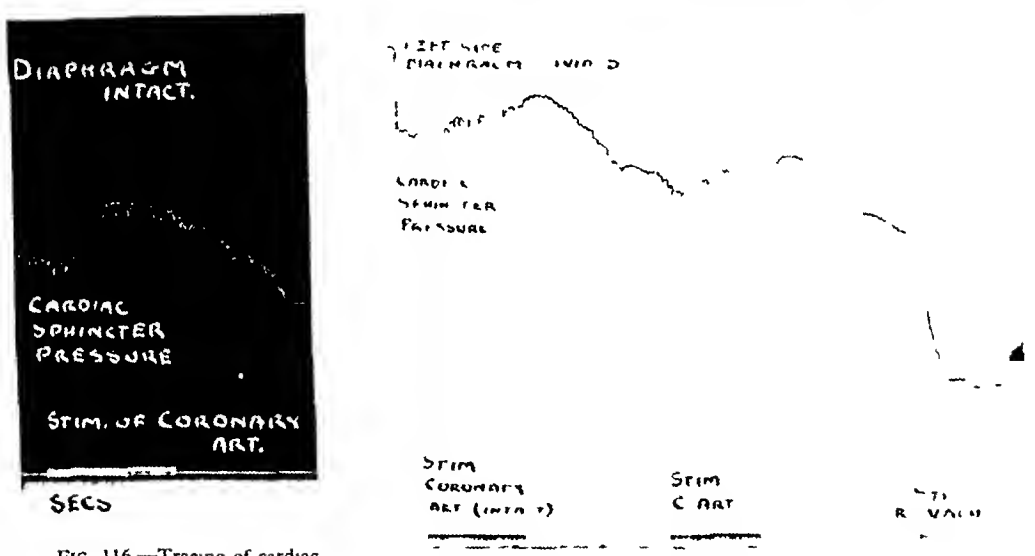


FIG 116—Tracing of cardiac sphincter pressure, showing contraction on stimulation of sympathetic nerves on the left gastric artery

FIG 118—Tracing of cardiac sphincter pressure, showing vagal inhibition and contraction on sympathetic stimulation with the diaphragm divided



FIG 117—Tracing of cardiac sphincter pressure, showing contraction on stimulation of the peripheral

surface of the œsophagus high in the thorax to interrupt those fibres descending in the plexus from the right recurrent laryngeal, and division of the left vagus in the neck, then the condition shown in *Fig 123* is produced. It shows enormous dilatation and an obstruction sufficiently severe to cause death.

Bilateral Excision of Stellate Ganglia—Increased peristalsis is observed in the middle and lower thirds. The sphincter is normal. There is marked hypertonicity in the upper third (striped muscular zone) (*Fig 124*).

Bilateral Excision of the Thoracic Sympathetic Chains—The third to tenth ganglia were excised in two stages through successive thoracotomies. *Fig 125* shows the condition six weeks later. There is a great increase in the peristaltic activity of the œsophagus, it being difficult to obtain a plate of the meal *in situ*. Digital pressure on the abdomen readily returns the meal from the stomach to the œsophagus, showing diminished sphincter tonus (*Fig 126*).

Bilateral Division of Vagi at Lung Root, with Bilateral Excision of Thoracic Sympathetic Chains (Ganglia 3-10)—Performed in two stages through successive thoracotomies. X-ray taken two months later. The œsophagus is at first somewhat dilated and shows decreased motility. The contraction at the diaphragm and intra-abdominal portion does not develop (*Fig 127*). When the meal has entered the stomach it can be returned to the œsophagus by digital pressure. diminished tonus (*Fig 128*). After some weeks there is a return of peristaltic activity and the tonus of the sphincter is regained, but obstruction at this region does not occur, the sphincter relaxing readily in the course of peristalsis as in the normal œsophagus.

Excision of Sympathetic Fibres surrounding the Celiac Axis Artery—The sphincter is completely patulous and cannot support the meal in the vertical position (*Fig 129*). The meal flows back passively into the œsophagus when the animal is placed in the horizontal position (*Fig 130*). After some months there is some recovery of tonus, but digital pressure still causes the return of the meal to the thoracic œsophagus.

Division of the Coronary Artery of the Stomach—Changes similar to the above, but less marked, the sphincter being in diminished tonus but not patulous.

Celiac Peri-arterial Sympathectomy in an Animal previously treated by Bilateral Division of the Vagi—*Fig 131* shows the condition of the œsophagus in an animal where both vagi had been divided below the lung root seven months previously. It had been vomiting its food for one week. *Fig 132* shows the condition after peri-arterial sympathectomy, the food passes into the stomach without any obstruction. The sphincter is shown in a relaxed condition. There is increased peristalsis of the entire lower third of the œsophagus.

DISCUSSION

It appears that the type of response obtained from electrical stimulation of the extrinsic nerves passing to the thoracic œsophagus depends upon the nature of the muscle content of the region under examination.

Thus in cats the upper third consists of striped muscle, and in it was obtained the type of response elicited by Kure and his co-workers²³ from the entire œsophagus in dogs. The striped muscle is prolonged throughout the entire extent



FIG 119 —X-ray of œsophagus of normal cat, showing no contraction at cardiac sphincter



FIG 120 —X-ray of normal cat, showing no return of the meal from digital pressure on the stomach contents



FIG 121 —X-ray of œsophagus following bilateral vagotomy, showing contraction at the cardiac sphincter



FIG 122 —X-ray following bilateral vagotomy, showing no return of the meal from digital pressure on the stomach contents



FIG 123 —X-ray following complete bilateral vagotomy, showing enormous dilatation and complete obstruction at the cardiac sphincter



FIG 124 —X-ray following bilateral excision of stellate ganglia showing hypertonicity of upper third and increased peristalsis. The sphincter was normal



FIG 125—X-ray following bilateral excision of thoracic chains, showing increased tonus and motility



FIG 126—X-ray following bilateral excision of thoracic chains, showing the return of the meal from stomach to oesophagus



FIG 127—X-ray following bilateral vagotomy and thoracic sympathectomy showing absence of contraction at the cardiac sphincter (Cf Fig 121)



FIG 128—X-ray following bilateral vagotomy and thoracic sympathectomy, showing return of the meal from stomach to oesophagus



FIG 129—X-ray following coeliac sympathectomy, showing that the meal cannot be supported in the vertical position



FIG 130—X-ray following coeliac sympathectomy, showing passive return of the meal to the oesophagus in the horizontal position

of the œsophagus in dogs (Arey and Tremaine³⁵) Further, the influence on prolonged stimulation is somewhat suggestive of the results obtained by Orbeli³⁶ upon the influence of sympathetic stimulation on fatigue in skeletal muscle, though differing from this reaction in its immediate and sudden effect

In the lower third the types of response elicited are those which are associated with the influence of extrinsic nerves upon unstriated muscle elsewhere in the gut—i.e., increased tonus and motility on vagal stimulation and inhibition of motility on sympathetic stimulation In this region in cats the muscle is predominantly unstriated The responses also showed a marked similarity to those obtained by Beattie and Sheehan³⁷ from the stomach in cats, which accorded with the observation of McSwiney³¹ that the musculature of the cardia gave a response to drugs which is 'gastric' in type rather than 'œsophageal' In the middle third variable effects were obtained

The results obtained from the inter-diaphragmatic and intra-abdominal portions of the œsophagus show the presence in this region of an intrinsic sphincter mechanism, since relaxation was obtained in all cases on vagal stimulation and contraction on sympathetic stimulation

The changes resulting in the œsophagus from excision of extrinsic nerve confirm the views expressed above Thus it has been shown that vagal stimulation caused an increase of tonus throughout the œsophagus, with increased motility of the unstriated area and relaxation of the cardiac sphincter Division of both vagi is followed by dilatation of the œsophagus and contraction of the sphincter The degree of dilatation and the severity of the obstruction are proportional to the extent of the denervation performed Immediately following this operation there was also a diminution of peristaltic activity, but this returned after some time as stated by Cannon—i.e., the unstriated area regained the power to respond to local stimuli by local contraction and peristalsis It is interesting to note that this activity is not accompanied by relaxation of the sphincter, which remains in a condition of increased tonus It has been stated elsewhere that this obstruction is only temporary in nature—with this we cannot agree *Fig 121* shows the condition in a cat four months after operation *Fig 131* is taken from the same animal three months later, and in this case the obstruction has increased in severity despite the increase in peristalsis

Stimulation of the sympathetic has been shown to excite certain effects upon the striped muscle of the œsophagus—diminution in the peristaltic activity of the lower third and contraction of the cardiac sphincter These views again receive confirmation from the results of the operations quoted above Thus bilateral excision of the stellate ganglia is followed by increased peristalsis in the middle third of the œsophagus Bilateral excision of the thoracic chains is followed by increased peristalsis and diminished sphincter tonus, and coeliac peri-arterial sympathectomy results in diminution of sphincter tonus

We were unable to see any sign of the peculiar retropericardial obstruction described by Kure²¹ following bilateral excision of the stellate ganglia, except in so far as hypertonicity of the upper two-thirds caused narrowing of the lumen

When bilateral vagotomy and excision of the sympathetic chains has been performed, the lower third of the œsophagus has been completely denervated, for the sympathetic supply to the sphincter must take its origin from this source, by whatever route it reaches its destination In such a case the effects at first observed

appear to be a combination of the above. There is a diminution in tonus and motility of the œsophagus and diminished sphincter tonus. The obstruction which results at this region when the vagi are alone divided does not occur. Later there is a return of peristalsis in the lower third, but this peristalsis is accompanied by relaxation of the sphincter, which does not occur when the vagi alone are divided.

As regards the possibility of the vagus exciting both motor and inhibitory action upon the sphincter, this is not disputed. It is well known that many nerves contain antagonistic fibres which may be selectively stimulated by suitable means. In these experiments contraction occurring at the cardiac sphincter on vagal stimulation has never been observed, but the experiments have in all cases been conducted with high frequency and in animals in the fasting condition. The predominant action of the vagus upon the cardia is inhibitory. In all cases vagus excision has been followed by contraction at the sphincter, and sympathetic excision by diminished tonus.



FIG 131—X-ray showing late result of double vagotomy. The obstruction at the sphincter has increased in severity (Cf Fig 121)



FIG 132—X-ray showing relief of the obstruction at the sphincter following coeliac sympathectomy (Cf Fig 131)

The bearing of these results upon the etiology of achalasia is interesting. Bilateral division of the vagi results in the clinical symptoms and X-ray appearances of achalasia, this effect being permanent and increasing in severity as time goes on. If the denervation is sufficiently severe, the symptoms are sufficient to cause death of the animal, and the post-mortem appearances of achalasia are produced. Fig 133 shows such a specimen at present in the Museum of the Royal College of Surgeons. It is the thorax of the animal whose radiographic appearances are shown in Fig 123. The œsophagus is enormously dilated—including the upper third—and 'filled the chest', simulating a mega-œsophagus. Rake³⁸ has shown that in many cases of achalasia show a degeneration of the cells of Auerbach's plexus. These cells Miss C J Hill³⁹ considered to be vagal relays, in which she is in agreement with Gaskell,²⁰ Kuntz,⁴⁰ and Abel.⁴¹

If, however, the sympathetic fibres are also removed, the obstruction and dilatation do not occur and the œsophagus becomes almost indistinguishable from the normal.

Further, if an animal is prepared so as to reproduce the clinical symptoms and

X-ray appearances of achalasia, subsequent sympathetic denervation of the cardiac sphincter results in a complete cessation of the symptoms, and radiograms show that food now enters the stomach. There is increased peristalsis of the lower third of the œsophagus and relaxation of the sphincter (*see Figs 131, 132*). There is no increase in the severity of the obstruction such as was observed by Kure in dogs²⁴. Food enters the stomach readily, whole meat being taken, whereas fluid only could be retained before operation. Vomiting ceases and the animal gains weight—in one case to the extent of 0.75 kilos in three months.

As regards the sympathetic innervation of the cardiac sphincter, dissection in human stillborns showed that the thoracic œsophagus derived its sympathetic innervation from two main sources: (1) A few direct branches from the thoracic sympathetic ganglia above the aortic arch, (2) The mesial branches of the ganglia via the peri-aortic plexus and œsophageal vessels. Immediately above the diaphragm no fibres to the œsophagus by either of these routes were seen. The experimental results show that stripping of the fibres surrounding the cœliac axis artery resulted in a patulous sphincter. In the cat these fibres are derived chiefly from the peri-aortic plexus, with some accessions from the cœliac ganglion. Some of these fibres reach the sphincter along the course of the left gastric artery, since division of this vessel results in a diminution but not complete loss of sphincter tonus, such as occurs after cœliac sympathectomy. The splanchnic nerves do not contribute to this supply to any appreciable extent, since no contraction of the sphincter could be produced by stimulation of these structures. This result received confirmation in the human from a case of cœliac sympathectomy performed by Mr J. B. Hume, Assistant Surgeon at St Bartholomew's Hospital, for gastric ulcer. Two years after operation the patient was X-rayed and the cardia specifically observed. The sphincter was seen to open abnormally readily, there being none of the usual delay at this region, although actual regurgitation could not be obtained.



FIG 133.—The thorax of a cat following bilateral vagotomy, showing œsophageal dilatation and obstruction at the sphincter sufficient to cause death (*Cf Fig 123*).

CONCLUSIONS

- 1 The œsophagus receives a sympathetic innervation
- 2 There is a true intrinsic sphincter at the cardia
- 3 Vagal stimulation causes tetanic contraction of the upper third of the œsophagus, which is composed of striped muscle. This contraction is augmented by sympathetic stimulation
- 4 Vagal stimulation causes increased tonus and motility of the lower third of the œsophagus, which is composed of plain muscle. The tonus and motility are inhibited by sympathetic stimulation

5 Bilateral vagal section, if complete, reproduces the appearances of achalasia of the cardia. Simultaneous removal of the sympathetic fibres prevents the onset of this obstruction. When the obstruction develops it can be relieved by section of the sympathetic supply to the sphincter.

6 The sympathetic supply of the cardiac sphincter is accessible as it passes to the sphincter along the course of the coeliac axis and left gastric arteries.

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REFERENCES

- ¹ OPENCHOWSKI, *Arch f Anat u Physiol*, 1889, 549
- ² MAY, *Jour of Physiol*, 1904, xxi, 266
- ³ LANGLEY, *Ibid*, 1899, xiii, 407
- ⁴ KOENNECKE, *Zeits f d ges exper Med*, 1922, xxviii, 384
- ⁵ RIFDER, *Deut Zeits f Chir*, 1929, ccxvii, 334
- ⁶ VEACH, *Amer Jour Physiol*, 1925, lxi, 229
- ⁷ CARLSON, BOYD, and PEARCEY, *Ibid*, 1922, lxi, 14
- ⁸ MCCREA, MCSWINEY, and STOPFORD, *Quart Jour Exper Physiol*, 1925, xv, 201
- ⁹ REID, *Edin Med and Surg Jour*, 1839, li, 274
- ¹⁰ BERNARD, *Comptes rend Soc de Biol*, 1849, l, 14
- ¹¹ SCHIFF, *Leçons sur la Physiologie de la Digestion*, 1868, 350
- ¹² KRONECKER and MELTZER, *Arch f Anat u Physiol*, Suppl, 1883, 328
- ¹³ D'ALMEIDA, *Comptes rend Soc de Biol*, 1929, cli, 407
- ¹⁴ KLEE, *Arch f d ges Physiol*, 1912, cxlv, 557
- ¹⁵ KREHL, *Arch f Anat u Physiol*, Suppl, 1892, 278
- ¹⁶ MCCREA, MCSWINEY, and STOPFORD, *Quart Jour Exper Physiol*, 1926, xvi, 195
- ¹⁷ CARLSON, *Amer Jour Physiol*, 1921, lxi, 299
- ¹⁸ CANNON, *Ibid*, 1907, xix, 436
- ¹⁹ JURICA, *Ibid*, 1926, lxxvii, 371
- ²⁰ GASKELL, *Involuntary Nervous System*, 1st ed, 1916, 73 London
- ²¹ VEACH, *Jour of Physiol*, 1925, lx, 457
- ²² PAGE-MAY, *Ibid*, 1904, xxi, 260
- ²³ KURÉ, *Arch f d ges Physiol*, 1929, ccxli, 367
- ²⁴ KURÉ, *Klin Woch*, 1929, viii, 491
- ²⁵ KUNTZ, *Autonomie Nervous System*, 1st ed, 1929, 188
- ²⁶ HOVELACQUE, *Anatomie des Nerfs*, 1st ed, 1927, 720
- ²⁷ TESTUT, *Traite d'Anatomie humaine*, 8th ed, 1930, iii, 408
- ²⁸ BRANDT, *Zeits f angewandte Anatomie und Konstitutionslehre*, 1919-20, 302
- ²⁹ HURST, *Quart Jour Med*, 1930, xxxi, 494
- ³⁰ POULTON and PAYNE, *Ibid*, 1923-4, xvii, 53
- ³¹ MCSWINEY, *Quart Jour Exper Physiol*, 1929, xix, 237
- ³² JACKSON, *Laryngoscope*, 1922, xxxii, 139
- ³³ MOSHER, *Ibid*, 348
- ³⁴ FULDE, *Deut Zeits f Chir*, 1934, ccxlii, 580
- ³⁵ AREY and TREMAINE, *Anat Record*, 1933, lvi, 315
- ³⁶ ORBELL, *Pavlov Jubilee Volume*, 1924, 411
- ³⁷ BEATTIE and SHEEHAN, *Jour of Physiol*, 1924, lxxvi, 218
- ³⁸ RAKE, *Guy's Hosp Rep*, 1927, lxxvii, 141
- ³⁹ HILL, *Trans Roy Soc, Sect B*, 1927, ccxv, 355
- ⁴⁰ KUNTZ, *Anat Record*, 1922, xliii, 193
- ⁴¹ ABEL, *Jour Anat and Physiol*, 1913, xlvii, 35

THE GROWTH OF PERIOSTEUM IN LONG BONES*

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THE growth of bones at one time appeared a simple matter. Like any other structure in the body they were believed to increase in length and thickness by what may be called *interstitial growth*—a process whereby the increase in size is attained by the growth of each small fragment in length and thickness to a varying extent. The femur of a child bears a close resemblance to the femur of an adult, the astragalus of a child to the astragalus of an adult. The theory that the growth is interstitial afforded a completely satisfactory explanation in each case.

This simple and satisfactory view was shattered by the experiments of John Hunter, who placed pellets at measured intervals in long bones and found that with the growth of the bone the pellets remained the same distance apart. This fact, combined with histological observations by Goodsir of the growing cartilaginous disc near the ends of the bone, established the view that long bones grow in length by linear proliferation of the cartilaginous disc, the so-called *epiphysial* or *diaphysial line*. As a result of the added complication of this growth disc, many problems of varying importance have been introduced into the puzzle of bone growth.

If a bone grows more or less equally at each end, the original shaft of the child must lie buried in the middle third of the shaft of the adult. If, as has hitherto been assumed, the periosteum also grows at its extremities, it is necessary to find some explanation of the fact that muscle attachments in the adult are not crowded into the middle third of the bone but are arranged in the same relative positions as in the child.

The relation of periosteum to bone growth has been much discussed, but no direct or at any rate conclusive observations have been made on the growth of periosteum itself. One suggestion that presents itself is that no matter how complicated are the processes that obtain in the interior of the bone, periosteum on the outside might conceivably grow interstitially. From an external aspect the growth process in long bones would then bear a close resemblance to that in short or irregular bones (e.g., the carpal and tarsal bones).

The object of the present experiments was to throw light on the behaviour of periosteum in the growth of long bones. It was felt that an understanding of this might help to explain the sliding of muscle insertion and perhaps throw some light on the vexed question of the relation of periosteum to bone limitation and modelling. (The question of the exact constitution of periosteum is left on one side, as it will be seen that our experiments only deal with the fibrous portion.)

* From the Courtauld Institute of Biochemistry

Rabbits were used for all the experiments. They were usually about six weeks old and were killed after a further six to nine weeks for examination. In every case the tibia of the hind legs was taken as the experimental bone. The insertions of the internal lateral ligament of the knee and of the anterior annular ligament of the ankle were chosen as clearly defined points of attachment of soft tissues to bone.

The experiments may be divided into four groups.

Group A.—The first series of experiments were carried out along the original lines of Hunter. The details of these experiments were as follows (*Rabbit 2, Fig 134*) —

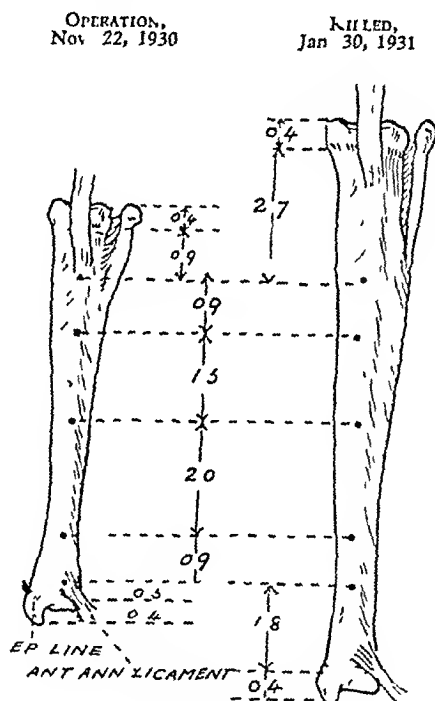


FIG 134—*Rabbit 2*
• = Wire pins

Silver wire pins were placed in holes drilled through the whole thickness of the bone at measured points which included among others those of the attachments of the internal lateral ligament of the knee and of the anterior annular ligament of the ankle. After nine weeks' growth it is seen that the pins remained the same distance apart, but the insertions of the ligaments had moved considerably from their original sites, indicated by pins. Pinning down the ligaments to the bone has made no difference to this movement.

The results of these experiments are in agreement with those of Hunter. No interstitial growth of bone has taken place. In addition, however, it is to be noted that the attachments of certain ligaments have moved along the bone. Reflection shows that the procedure throws no light on what happened to the periosteum either during the experiments or under natural conditions. Just as the ligaments travel along the bone, so conceivably may the periosteum itself. It may have

grown interstitially or have grown from the end only, or have been prevented from growing by being attached to the underlying bone by the pins

Group B—It was necessary to mark the periosteum without attaching it to the underlying bone so as to permit it to grow naturally. This presented some difficulty. At first an attempt was made to differentiate growth of periosteum from bone by threading wire loops through the periosteum. It was then found that the part of the loop on the deep surface became incorporated with the bone and remained fixed, although rotation of the loop sometimes occurred, it was obvious that this was not a satisfactory method. Various devices were tried and discarded. Finally, acting on the suggestion of Dr Scarff, Indian ink was tried, and proved to be quite satisfactory.

The bone was exposed with the usual surgical aseptic technique and the periosteum carefully dried with swabs. At suitable intervals marks of Indian ink

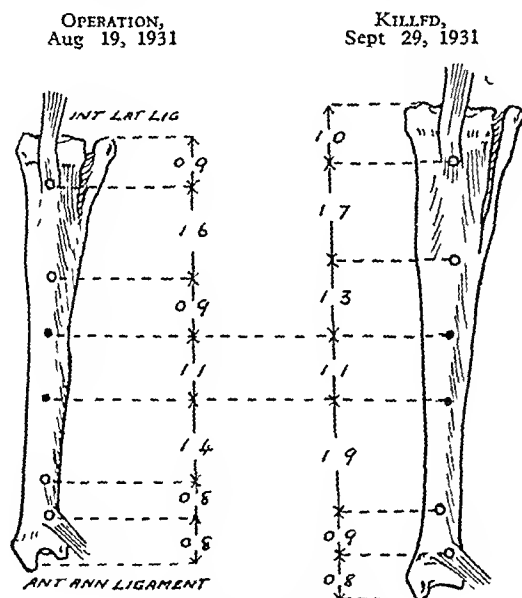


FIG 135—Rabbit 6
● = Wire pins ○ = Indian ink

were then made superficially in the substance of the periosteum with a mapping pen. Dividers were used for making all measurements, which are recorded in centimetres. At the end of two months the marks, with a few exceptions, were still clearly visible, although they had become slightly fainter and more diffuse. Owing to the impossibility of making extremely small marks, the measurement between any pair of marks can only be considered accurate to the nearest millimetre.

Metal pins placed through the diaphysis of the bone remain unchanged in position and can be regarded as fixed points from which to make future measurements. Two pins were placed through the bone to act as fixed points and ink marks were made on the periosteum and ligaments. If the periosteum grew only by increments from its ends, then the positions of the ink marks relative to the pins should remain unchanged. That this does not occur is very clearly shown by Rabbit 6 (Fig 135).

Clear evidence was thus obtained that interstitial growth of periosteum takes place. Ink marks placed in the ligaments at their insertion were found still to remain at the junction of ligament and periosteum.

Group C.—Further experiments showed that the periosteum adjoining the ligamentous attachment changed its position with that of the ligament itself. Ink marks were made on the ligaments and the periosteum at exactly the same level. The relative positions of these two marks were unchanged when the animals were

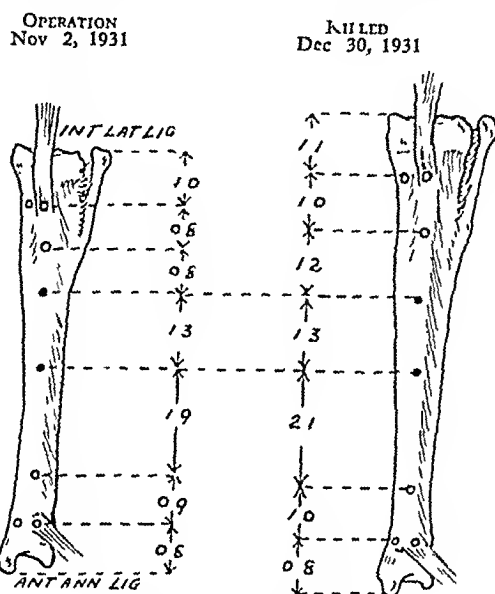


FIG 136—Rabbit 10
● = Wire pins ○ = Indian ink

killed (Rabbit 10, Fig 136). It is therefore unlikely that the drag of the shifting of ligament insertions plays much part in the separation of given points in the periosteum.

It has thus been shown that the insertion of a ligament always preserves the same relationship to the fragment of periosteum to which it was originally attached. In other words, the two halves of a mark which at any period indicates the junction of the periosteum with any given ligament remain undivorced during growth of the bone, and presumably the same also holds good for the relationship between periosteum and any muscle insertion.

It would have been of interest in this connection to observe any change in the position of the lower tibio-fibular synostosis relative to the points fixed by the pins. However, it was not thought advisable to attempt to make this observation at the same time from fear of damaging periosteal growth.

It is now necessary to consider briefly how the ligamentous insertion changes its position. If the ligament is merely attached to the periosteum itself, no difficulty arises, as the ligament simply moves with the growth of its related periosteum. The solidity with the bone beneath must then be brought about by the rapid fixation of fresh periosteal perforating fibres formed in the subperiosteal plane in response to the stress on the slowly shifting insertion.

It is generally held, however, that "where tendons or ligaments are inserted into bone, the fibre bundles of the tendon are continued into the bone as perforating fibres, so that the attachment of tendon to bone is thus rendered very intimate" (Schafer¹) If this view is accepted, the perforating fibres must be supposed to elongate as the ligament shifts its position, for they cannot be supposed to re-insert themselves higher up the bone. The elongation may take place in the bone itself or in the part of the fibres which runs immediately under the periosteum. The latter suggestion is more likely. It may then be supposed that the freshly formed periosteal bone fixes the fibres in their slowly changing position. The subperiosteal changes are not affected by transfixation of the ligament to the bone by pins.

No matter what view of these fibres is taken, the effect produced is that the insertion of ligament and probably of muscle with the associated pits and ridges marking these insertions must actually travel subperiosteally along the bone as it grows.

Group D.—Finally an attempt was made to discover if the increase in length of the periosteum took place by regular interstitial growth—that is to say, as evenly as a piece of elastic stretches—or if it were more irregular. To this end a series

Operation Killed	RABBIT 15 Jan 28, 1932 March 9 1932		RABBIT 14 Jan 21, 1932 March 9, 1932		RABBIT 13 Jan 20, 1932 March 9, 1932	
EP LINE	04	04	04	04	03	03
LIGAMENT	05	05	05	07	05	08
	05	05	05	} 11	05	06
	05	06	05		05	06
	05	06	05	07	05	06
	05	} 13	05	06		
	05		05	05		
	05	07	05	05		
	05	06	05	08	05	07
	05	07	05	09	05	07
	05	06	05	07	05	07
LIGAMENT	05	06	05	06	05	08
EP LINE	05	05	04	04	04	06
	04	04	04	04	03	03
Total length	68	80	67	83	50	67

FIG 137

of ink marks was made on the periosteum of the entire length of the shaft at intervals of 0.5 cm. After growth it was found that, with few exceptions, each interval was increased in size, although not all to the same extent. Occasionally isolated ink spots could not be found. (Fig 137)

SUMMARY AND CONCLUSIONS

1 The growth of the periosteum of the tibia of young rabbits was observed by means of marks of Indian ink

2 It was found that periosteal growth did not occur as in the underlying bone by increments from the epiphysial lines, but by interstitial growth throughout its length

3 The limited number of observations that were carried out indicate that the interstitial growth is reasonably regular The results are recorded

4 The process of ligament and muscle sliding during normal growth of long bones, and possibly also the limiting of growth and modelling of the bone, are related to the interstitial growth of periosteum

We wish to express our indebtedness to Professor Dodds for the hospitality of the Courtauld Institute of Biochemistry, in which the work was carried out Our thanks are due to Mr E R E Spence, technician, for his assistance and care of the animals

REFERENCE

- ¹ SCHAFER, *Quain's Anatomy*, 1912, II, Part I, 151

*SHORT NOTES OF RARE OR OBSCURE CASES***FALLOPIAN TUBE IN THE MALE :****DISCOVERED WHILE OPERATING FOR FEMORAL HERNIA**

By J A BATY

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A MINER, aged 29, attended the Out-patient Clinic of the Cumberland Infirmary, complaining of "rupture" in the right groin. He stated the rupture had been present since birth. The swelling always disappeared spontaneously when he lay down. There had never been symptoms of threatened obstruction. His own doctor had attended him a few months previously for an attack of left-sided epididymitis which lasted about a fortnight. There has been a transitory yellow, opaque, sticky discharge, but no other urinary symptom. No evidence of gonococcal infection had been discovered. The patient had been married over a year. Marital relations were apparently normal, but birth control had been practised and there was no child.

ON EXAMINATION —The patient was well developed and had a rather pugilistic type of face. All the secondary male characteristics were in evidence. No testis could be felt in the right side of the scrotum, inguinal canal, or femoral triangle. There was a femoral hernia which could easily be reduced. On the left side the testis was of average size with a definite corpus testis and epididymis, and lay in the scrotal sac. The cord was normal and there was no detectable hernia. Per rectum, the prostate was found to be very small, globular, and no larger than a pea.

OPERATION (Nov 30, 1933) —Operation for right femoral hernia was performed by Mr J N J Hartley. The inguinal canal was first explored. There was no sign of the testis, cord, or hernial sac in the canal. The femoral hernial sac was then dissected free and brought up into the inguinal canal. It was about the size of a thumb and when opened the neck readily admitted the index finger. A search was made for an undescended testis, but no testis could be felt by the finger. While retracting the neck of the sac, the free extremity of a tube appeared at the inferomedial margin. This was gently brought out, and to our surprise it had all the appearance of a miniature Fallopian tube. By further traction the tube was seen to be continuous with a firm muscular structure about the size of the distal phalanx of the little finger. The opposite angle of this body could not be reached and it was therefore not determined whether a tube existed on the left side.

The mesosalpinx and the inner end of the tube were ligated, and the tube was removed for investigation. The neck of the sac was ligated by a purse-string suture applied from within and the hernial sac pursed up by a catgut suture. The femoral walls were narrowed by non-absorbable sutures and the wound was sutured.

DESCRIPTION OF THE TUBE—The excised tube is about 3 in in length, and, as will be seen from the accompanying sketch (*Fig 138*), possesses a fimbriated extremity with a distinct ostium. The tube is lying along the free upper border

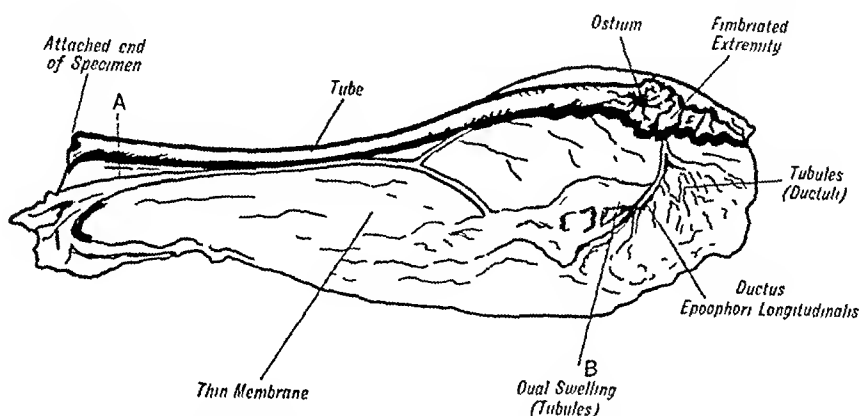


FIG 138—Specimen of Fallopian tube removed from male patient. Drawing of specimen from posterior aspect. A and B indicate source of sections for microphotographs.

of a thin membrane on the posterior aspect of which a number of rudimentary structures can be seen. Passing out from under cover of the fimbriated end of the tube is a definite ridge which divides into two main stems forming a triangular



FIG 139—Section taken from A in *Fig 138*. Fallopian tube. Low power view showing structure. For details see text.

area occupied by smaller radiating ridges. These, at the periphery, anastomose. Just above the medial main ridge there is a flattened boss about $\frac{1}{2}$ in by $\frac{1}{4}$ in and below the latter are further ridges, branching and rejoining. From the medial end

of this system a single ridge arches up towards the tube and for a short distance runs parallel along the latter's lower border. Separation of these structures again occurs at a point about $\frac{3}{4}$ in from the attached end of the specimen.

Histological investigation (Fig 139) shows the tube to possess a tunica serosa separated by a tunica adventitia of loose connective tissue from a tunica muscularis. The latter is composed of two strata of smooth muscle fibres, the outer longitudinally arranged and the inner circularly. The submucosal layer of embryonic fibroblastic cells supports the mucous membrane, which is thrown into a number of longitudinal folds. The lining epithelium consists of columnar cells with large, pale central nuclei showing a fine chromatin network. The free surface presents small protoplasmic protusions which are definitely cilia.

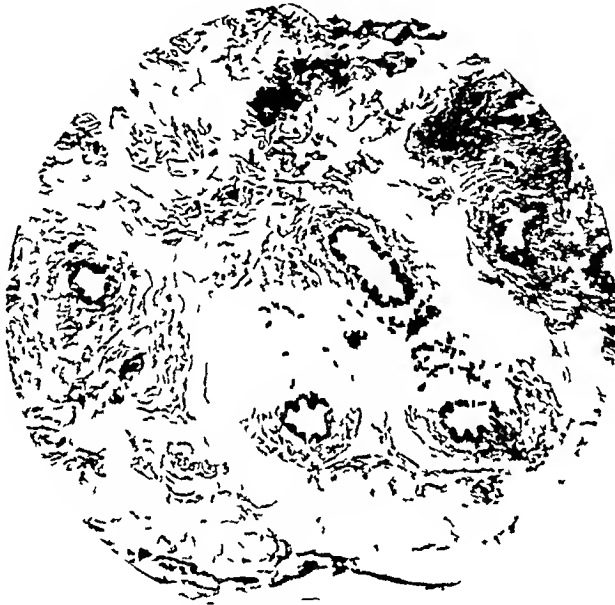


FIG 140 —Section taken from B in Fig 138. Tubules. Low-power view showing structure of oval swelling below fimbriated extremity of tube.

A section taken to include the ridges and oval swelling in the membrane below the fimbriated end of the tube reveals no evidence of ovarian or testicular tissue. For the greater part this area is composed of small tubules lined by ciliated epithelium and held in apposition by loose connective tissue (Fig 140).

COMMENTARY

There can be no doubt that in this patient there existed a diminutive but well-developed Fallopian tube. The Mullerian element on the right side has become dominant. There are vestiges of the Wolffian derivative, and these are more in evidence than in the normal female. The epoophoron with its ductus epoophori longitudinalis can readily be distinguished. The ductuli correspond to the efferent ductules of the testis, but no spermatocytic cells are present. The ductus epoophori longitudinalis is the remains of the Wolffian duct and is the homologue of the vas in the male and the duct of Gartner in the female.

The embryological explanation for the developmental abnormality can only be conjectured. Undoubtedly sex differentiation rests upon chromosomal constitution. It is determined at the moment of union between sperm and ovum according to whether the sperm carries the X or Y chromosome. Before sex differentiation becomes reinforced by endocrine activity, the chromosomal influence appears so weak that the developing embryo passes through an indifferent phase, structures proper to both male and female making their appearance. This phase is followed by a masculinizing tendency in both sexes. In the female a testicular rudiment appears in the rete of the ovary, the investing ovarian cortex becoming the real ovary. In the male the tendency is evidenced by the earlier differentiation of the testis. It is held that the testis is a monosexual organ, whilst the ovary is bisexual, and that sex reversal can only occur in the female. Despite the recent work on accidental and experimental sex reversal, it would be hazardous to assume in the human subject with a testis so well developed that the chromosomal content was other than male. The rudimentary excretory apparatus when the sex gland is absent calls for no comment. It is not clear why the Mullerian element should develop to such an extent in a male, for mere absence of one testis does not lead to unilateral preponderance of female organs. Until there is a more exact knowledge of the sexual hormones and their actions, the true explanation of this case will remain in doubt.

A RARE ABNORMALITY OF THE HEPATIC ARTERY

By G A G MITCHELL

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ABNORMALITIES of the cystic and hepatic arteries and of the biliary apparatus are well recognized and their surgical importance has been frequently emphasized, but it is doubtful if many surgeons realize that the presence of an abnormal cystic artery not infrequently indicates an abnormality of the parent hepatic stem. The hepatic artery and its branches conform to the text-book description in only 50 to 55 per cent of cases. In the remainder abnormalities of the most varied character may be encountered, some relatively common, others comparatively rare. The discovery of one of the latter in a male subject aged 63 prompts me to give the following details.

The abdominal aorta was normal and the branches came off in the usual situations, but the coeliac axis was smaller and the superior mesenteric artery larger than usual. The coronary and splenic branches of the coeliac axis conformed to the usual arrangement, but the hepatic artery was very small, and its only branch was a tiny twig to the pyloric region of the stomach, it gave off no other macroscopic branches before dividing into minute vessels which entered the liver.

Arising from the superior mesenteric artery in front of the uncinate process of the pancreas was a vessel which was as large as the continuation of the artery itself. It ran upwards in the *anterior* groove between the duodenum and the head of the pancreas, then behind the first part of the duodenum, and finally in the

free margin of the lesser omentum to the right of the common bile-duct, which thus had an artery on each side and the portal vein behind it. This abnormal branch gave origin to the middle colic, right gastro-epiploic, pyloric, and cystic arteries (*Fig 141*), and supplied also those structures usually supplied by the pancreatico-duodenal vessels. Finally it entered the portal fissure, its two terminal branches lying anterior to the hepatic ducts. The cystic artery lay below and to the right of the cystic duct. The biliary apparatus and the other viscera were normal, and there was no indication that the abnormal artery was produced by

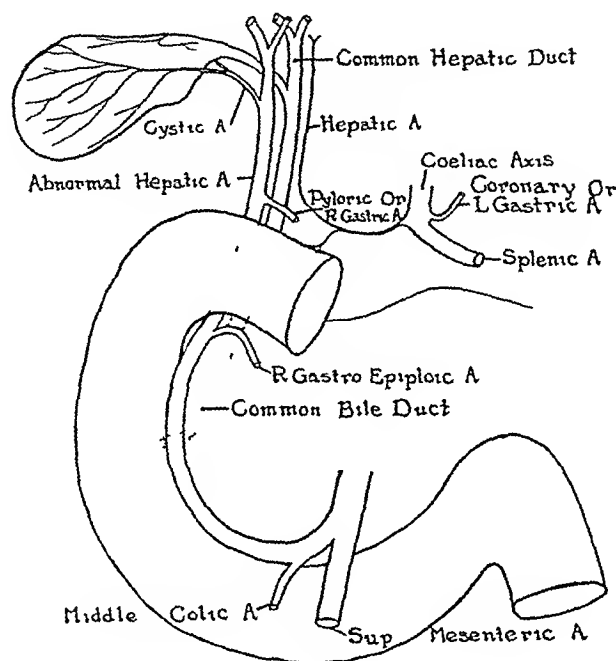


FIG 141—Abnormality of the hepatic artery

post-natal enlargement of the anastomoses normally present between radicles of the pancreatico-duodenal arteries.

The appearances suggested that the major part of the liver, the biliary apparatus, the pyloric end of the stomach, the greater part of the duodenum, the head of the pancreas, and the transverse colon received their arterial blood-supply through this curious vessel. Any injury, operative or otherwise, to such a vessel would be fraught with the gravest consequences, and from its situation it would be endangered in most operations in this region.

In his exhaustive "*Essai sur l'Anatomie et la Medecine operateire du Tronc coeliaque et de ses Branches*" Rio-Branco discusses abnormalities of the hepatic artery, but of the large number of abnormalities he describes there is not one exactly similar to the above, the nearest approach to it is a case reported by Wiart. Moreover, in the cases described in the literature I have not found any in which so many structures were so largely dependent on the abnormal artery.

The origin of an abnormal hepatic artery from the superior mesenteric artery is a well-known possibility, but almost invariably it arises near the beginning of

the superior mesenteric, *behind* the pancreas, and then passes upwards and to the right, behind the first part of the duodenum, towards the free margin of the lesser omentum, where it may lie to the right or to the left of the common bile-duct or posterior to it. In one case described by Corning the abnormal vessel, although arising behind the pancreas, passed *anterior* to the first part of the duodenum. In most of these abnormal cases another hepatic artery exists, arising as usual from the cœliac axis or directly from the abdominal aorta, or less commonly from the coronary or splenic arteries. This artery may be larger or smaller than the one arising from the superior mesenteric, and it is sometimes called the 'left' or 'superior' hepatic artery, in contradistinction to the other, which is described as the 'right' or 'inferior' or 'accessory' hepatic artery, the term 'accessory', however, is misleading, because in many instances the right or inferior artery is the larger of the two. They are designated 'right' and 'left' because in their final distribution they usually conform more or less closely to the right and left terminal branches of the normal hepatic artery, but Corning, Leriche, and others record cases where the so-called left or superior hepatic artery ended in the right lobe of the liver. In other cases three hepatic arteries are present, which frequently arise as follows: the first from the coronary or less often from the pyloric artery, the second from the cœliac axis or directly from the abdominal aorta, and the third from the superior mesenteric artery or from a combined cœliaco-mesenteric trunk. Isolated cases have been recorded of abnormal hepatics arising from, or in conjunction with, the renal or inferior phrenic arteries, and from the inferior mesenteric artery.

In 338 subjects examined by Leriche and Villemain, Sousloff, Rossi, and Cova, and by Rio-Branco, the hepatic artery arose from the cœliac axis in 315 cases, from a combined cœliaco-mesenteric trunk in 4 cases, from the superior mesenteric artery in 14 cases, and directly from the aorta in 5 cases. Thus in 4 per cent of cases an abnormal hepatic artery may arise from the superior mesenteric artery. It is worth emphasizing again that in the great majority of these cases the artery originates and pursues a relatively safe course behind the pancreas and first part of the duodenum, but in the specimen described above the abnormal hepatic arose anterior to the pancreas and pursued a totally different course. The latter abnormality is comparatively rare, and from a surgical point of view much more dangerous.

I am indebted to Professor Low, Department of Anatomy, Aberdeen University, for kind permission to publish this case.

BIBLIOGRAPHY

- CORNING, *Lehrbuch der topographischen Anatomie*, 2nd ed., 421. Wiesbaden. Bergmann.
 FLINT, *Brit Jour Surg*, 1923, x, 509.
 GRAHAM and CANNELL, *Ibid*, 1933, xx, 566.
 LERICHE and VILLEMINE, *Bull et Mem Soc anat de Paris*, 1907, lxxxii, 224, *Bibliogr anat*, 1907, xvi, 116.
 RAIGORODSKY, *Zeits f d gesamte Anat*, 1928, lxxxvi, 698.
 RIO-BRANCO, *These de Paris*, 1912, clxviii, 411.
 ROSSI and COVA, *Arch ital di Anat e di Embriol*, 1911, fasc 2, 485, fasc 3, 566.
 WIART, *Thèse de Paris*, 1899, 29.

DISLOCATION OF THE ELBOW WITH RUPTURE OF THE BRACHIAL ARTERY

BY RALPH MARNHAM

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ON May 16, 1933, Mrs M, a woman aged 40, fell from a step-ladder, a distance of some four feet. She was able to describe the accident accurately. She fell on to her left hand with the arm extended, the forearm fully supinated, and the hand dorsiflexed.

ON EXAMINATION—When seen an hour after the accident she complained of pain at the elbow-joint and tingling of all her fingers. There was extreme swelling in the region of the elbow-joint, and in the absence of an X-ray it was impossible to assess the exact nature of her injury. She had no signs of damage to nerves, but the radial pulse was absent.

OPERATION—It was decided that she should be operated upon immediately. An incision 4 in. long was made over the anterior aspect of the internal epicondyle. On dividing the skin a large blood-clot was immediately evacuated. When the edges of the incision were retracted only two structures were seen running across the front of the elbow-joint, the one was the tendon of the biceps, the other the median nerve. All the muscles had been torn off the internal epicondyle and the brachial artery had been divided about $\frac{1}{2}$ in. above its bifurcation. There was a firm clot about $\frac{1}{2}$ in. long in the proximal lumen. The radius and ulna were dislocated backwards without any fracture, this was subsequently confirmed by an X-ray examination. The dislocation was reduced and the proximal end of the brachial artery ligatured, the distal end of the brachial artery had retracted, but was found and ligatured. The torn muscle was approximated by mattress sutures. After the skin incision had been closed, the arm and hand were covered with several layers of cotton-wool and kept warm with hot bottles. The arm was placed on a pillow in about 20° of flexion and in full pronation.

SUBSEQUENT PROGRESS—The next day the arm and hand were warm and sweating. The radial pulse had reappeared by the end of three weeks. By the end of four months extension of the elbow-joint was limited by about 10°, otherwise movements were full. There was no noticeable loss of power in flexing the forearm. The patient never had massage or passive movements.

The points of interest in this case are (1) The rarity of the condition—I have been able to discover only one other case recorded in the literature¹ for the last thirteen years, (2) That with trauma sufficient to rupture not only the brachial artery but also the flexor muscles, the nerves, particularly the median, and the collateral circulation should have remained undamaged.

REFERENCE

¹ *Jour de Med de Bordeaux*, 1917, July, 522

LARGE PREVERTEBRAL HÆMATOMA CAUSING PARALYSIS OF THE RECURRENT LARYNGEAL NERVE

By CECIL P G WAKELEY
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RICHARD S, age 22, a tyre fitter, was admitted to King's College Hospital on Nov 21, 1933, complaining of a very swollen neck and difficulty in swallowing and speaking. Half an hour before admission, while trying to lever a motor tyre on



FIG 142 —Skiagram showing foreign body in the 7th cervical vertebra

to the wheel of a lorry, a small piece of the tyre lever was chipped off and pierced his neck. The wound of entry was very small and there was but little bleeding.

On examination a small wound was seen about $\frac{1}{4}$ in in diameter and $1\frac{1}{2}$ in above the left sternoclavicular joint. A lateral X-ray revealed a foreign body about $\frac{1}{4}$ in long situated in the anterior part of the body of the 7th cervical vertebra,

a little to the left of the mid-line. The skiagram also shows the track of the piece of metal as it passed through the fascia of the neck (*Fig 142*)

Examination of the interior of the larynx revealed paralysis of the left vocal cord

The patient was admitted to hospital as his condition did not improve. The next day the wound of entrance was excised and the wound explored. With a bullet probe the piece of metal could be felt firmly embedded in the vertebra, but as the exploration caused considerable bleeding it was deemed wise not to proceed further. The large hæmatoma gradually subsided and the swelling ceased to be painful. On examination with the œsophagoscope a large bulge could be seen on the posterior pharyngeal wall. The paralysis of the left recurrent laryngeal nerve gradually cleared up, and by the end of three weeks there was no sign of any involvement of this nerve, and the patient was discharged.

This case is of interest because of the curious nature of the injury and the large size of the prevertebral hæmatoma which caused paralysis of the recurrent laryngeal nerve. The piece of metal must have taken a course very near the common carotid artery, as it must have pierced the carotid sheath.

BILIARY CYST OF THE LIVER*

By E. ROCK CARLING

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B. J., female, aged 28, was admitted to Westminster Hospital in July, 1927. For six years she had observed an enlargement of the veins on the right side of the trunk, and latterly they had become very much larger; she was troubled by a dull aching pain on the right side and had consulted Dr. Alment, of Abbot's Langley.

ON EXAMINATION—The veins extended from the umbilical region up over the chest, mainly towards the right breast and axilla, but partly also over the lower part of the sternum. The blood flowed upwards in the umbilical, superior epigastric, lateral axillary, and mammary vessels. Close to the umbilicus a definite thrill was palpable in the dilated veins. The liver projected about two finger-breadths below the costal margin and could be felt on careful palpation.

Upon radiographic examination the diaphragm was seen raised to the level of the 3rd rib. There was no marked displacement of the heart. The radiologist suggested that there might be a tumour between the liver and diaphragm.

The temperature ran from 98° to 99° in the evening; the urine was normal; bowels freely open. Hæmoglobin, 75 per cent; red blood-cells, 4,770,000; white blood-cells, 5600; Wassermann reaction, negative; cholesterol, 110 mgrm per 100 c.c.; bilirubin, 0.1 units; lævulose tolerance, normal. No hæmorrhoids. No symptoms of importance.

OPERATION—After the case had been shown at 'consultations' it was decided to explore. The right lobe of the liver was occupied by a large cyst which came

* For a bibliography of non-parasitic cysts of the liver, see the article by Ackman and Rhea, *BRITISH JOURNAL OF SURGERY*, 1931, viii, 648.

to the hepatic surface just lateral to the gall-bladder and was there thin-walled. It was incised, and a large quantity (2½ pints) of golden yellow liquid with quantities of cholesterol evacuated. Bile pigments and salts were present. After emptying, the cavity easily admitted the whole hand and wrist, so that it was possible to feel and compress the inferior vena cava at the back of the liver. There was no 'cyst wall' separable from liver substance over the greater part of the parietal area and very little collapse of the liver substance, so an anastomosis was made with the greater part of the adjacent gall-bladder, using the same technique as for joining two hollow viscera.

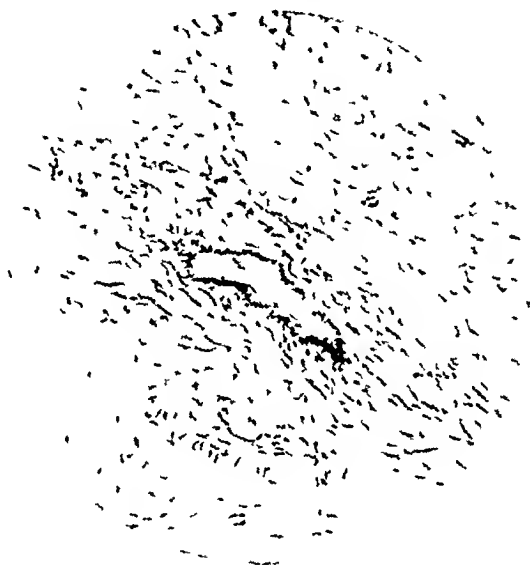


FIG 143—Histology of the biliary cyst of the liver

Recovery was without other incident than the formation of a sinus that healed after removal of a ligature. In a month's time the diaphragm, though still at the level of the 4th rib, was now freely mobile, whereas before the operation it had been stationary. The veins were perceptibly smaller two days after operation and gradually disappeared. There was a slight excess of cholesterol in the faeces. The cyst wall showed a hyaline fibrous matrix with very thick walls, with small spaces lined by cuboidal cells resembling those of the biliary ducts (*Fig 143*).

LARGE SOLITARY CYST OF RENAL ORIGIN

By E ROCK CARLING

SURGEON, WESTMINSTER HOSPITAL

In the year 1911 Mrs E B, aged 47, was sent for consultation by Dr Anderson, of Alleyn Park. She was preceded into the room by her husband, who walked backwards, supporting his wife's abdomen with both hands. Without such help she could scarcely stand, and certainly not walk, so great was the bulk and weight of the cyst.

Her general health was fairly good and she had no symptoms other than those due to the size of the abdomen. Micturition and the urine were normal. There was no jaundice. Though thin, she was not emaciated, she took food in small quantity but regularly, the bowels were regular, catamenia regular, but lately a little excessive.

She had allowed herself to suffer disabling discomfort, because, so she alleged, the most highly distinguished gynaecologist of that time, who had seen her in 1896, fifteen years before, had advised her never to allow any surgeon to operate upon her—thus although he had diagnosed an ovarian cyst.

ON EXAMINATION—The entire abdomen seemed to be filled with fluid, presumably contained within limiting walls, but it was impossible to identify any organ. The stomach and intestines seemed to be on the left side, but the only area of resonance obtainable was very small in extent, the pelvis was full of the 'cyst', which was thought (in view of the earlier diagnosis), although it was quite immobile, to be of ovarian origin.

OPERATION—An incision, which was meant to be paramedian, exposed the wall of the cyst without traversing the peritoneum. Upon incision twenty-five pints of fluid were collected and much was lost. The fluid was odourless and turbid. The turbidity was due in the main to cholesterol, which was present to the amount of 4 lb, there was also a considerable amount of squamous epithelium, the reaction was amphoteric, there was in this fluid no reaction for urea.

The entire peritoneal cavity and all the viscera had been pushed over to the extreme left. With the hand inside the cyst, the aorta and spinal column could be closely palpated. A renal origin seemed probable at this stage of the operation, though something that seemed to be the normal kidney could be palpated.

An attempt to enucleate the cyst resulted, whilst the dissection was still being conducted well above the collapsed abdominal wall, in damage to the posterior wall of the duodenum, fortunately not opening the mucosa. As much as possible of the cyst wall was excised, and the cavity then marsupialized.

SUBSEQUENT HISTORY—The discharge, three weeks later, had a specific gravity of 1030, effervesced with sodium hypobromite, contained squamous, pyriform, and spheroidal cells and triple phosphates, it did not digest albumen or form sugar from starch. The discharge ceased before the patient left hospital, but re-opened at times up to 1915, when she was lost sight of. In 1912 the patient returned to hospital for a hysterectomy for fibroids, which had increased in size with great rapidity in the intervening fifteen months.

Commentary.—It is possible that this was not a 'cyst' in the true sense, but a hydronephrosis into which a hæmorrhage had occurred, but the mass of renal substance palpated through the wall seemed normal, and at the time in question pyelograms were not in use.

The urine passed had a specific gravity of 1018 and contained oxalates, in the fluid from the sinus, which contained very small quantities of urea, there were triple phosphates, and the pathologists, Dr Hebb and Dr Braxton Hicks, believed that there was at least a connection with the renal pelvis. As some pararenal cysts have such a connection, this extreme example should probably be so classed.

MALIGNANT DISEASE STARTING IN A PHARYNGEAL POUCH

By DUNCAN C L FITZWILLIAMS

SENIOR SURGEON, ST MARY'S HOSPITAL

IN the BRITISH JOURNAL OF SURGERY for April, 1933, Capp and Dunhill published the details of a case of malignant disease which had started in a pharyngeal pouch before operation. Owing to the rarity of such a happening, perhaps the following case where malignant disease started in a pharyngeal pouch after operation may prove interesting.

W R H, a man aged 55, was admitted to the Freemasons Hospital in January, 1928.

HISTORY—For two to three years he had experienced difficulty in swallowing, which had gradually increased till he was unable to eat solids. His food in part regurgitated unchanged with no acid taste. There was no loss of weight.

ON EXAMINATION—He was a short man with a thick neck, there was fullness above each clavicle, but this could not be felt to increase on either side on eating.

Report of the X-ray examination The first mouthful swallowed was held up at the level of the top of the sternum and shows as a smooth rounded mass. The next mouthful passed fairly easily down the œsophagus, flowing past the upper portion of the previous swallow. These points indicate an œsophageal pouch.

FIRST OPERATION—At the operation I was assisted by my colleague, the late William Hill. An incision was made on the left side of the neck, the sternomastoid muscle was defined and drawn forward, the omohyoid was divided. A bougie was now passed into the pouch through the mouth, and the pouch defined, freed, and brought to the surface. It was very thin, consisting only of mucous membrane. Fine catgut sutures were inserted into the free end of the bag, which was displaced upwards and attached to the fibro-fatty tissue at the lower pole of the parotid underneath the superior thyroid vessels. The omohyoid was joined up and the wound closed with a drain. The sutures must have penetrated the wall of the pouch, as there was a slight discharge from the wound, and later some fluid escaped upon drinking. It is difficult to understand how sutures could be inserted into a tissue of such thinness without penetrating it.

SUBSEQUENT HISTORY—The patient left hospital in about three weeks and passed under the care of Dr Kirby, of Brighton, and although I heard of him from time to time I did not see him again for five years.

He was re-admitted to the Freemasons Hospital in March, 1933, with the following history. Since his operation a tiny sinus had persisted more or less all the time, healing and breaking down from time to time, but giving no trouble in any way. About four months previously other sinuses began to form, he also began to have trouble with the swallowing of solid food, and phlegm formed in his throat on eating.

On examination the skin on the left side of his neck was red, very thickened and indurated, and puckered into folds. In the folds the opening of six sinuses could be seen from which a little pus could be pressed. On probing the sinuses all seemed to converge upon one opening into the œsophagus.

SECOND OPERATION—An operation was performed, the sinuses were opened up, and most of the thickened skin was cut away after the manner of dealing with an anal fistula with multiple openings. The skin was so suspicious that it was sent to the pathologist for examination.

PATHOLOGICAL REPORT—Squamous-celled carcinoma was found in the skin and subcutaneous tissue. The growth is in large indurated masses with little keratinization. Mitotic figures are few in number.

On receiving this report radium was inserted into and around the wound and 6200 mgm-hrs were given. He developed a good reaction and the wound began to heal and was quite small when he left the hospital.

Two months later Dr Kirby wrote to me to say that he had been called to the patient one morning and found him gasping for breath. "There was definite stridor, and a swelling at the back of the pharynx, it looked like a retropharyngeal abscess." He was sent into hospital but it was decided not to operate, and he died the same evening.

REVIEWS AND NOTICES OF BOOKS

The Life of Sir Robert Jones By FREDERICK WATSON Medium 8vo Pp 327 Illus-
trated 1934 London Hodder & Stoughton Ltd 12s 6d net

THIS is a book written with perspicuity, sympathy, and love. The character of Sir Robert Jones is delineated with great exactness. His devotion to his life work, his love of humanity, suffering or otherwise, his generosity, tact, kindness, and sympathy are well shown and without a word too many.

After a brief account of Jones's boyhood days, we are introduced to that dour genius Hugh Owen Thomas, who gave him his first insight into the principles of orthopaedic surgery. Then we meet the grown man, whose appointment as Chief Surgeon to the Manchester Ship Canal gave him the opportunity of developing his powers of organization, a training which was to prepare him for the call of the War, where he showed himself a giant and did giant's work.

That Sir Robert Jones was careless in his work for advancing science is shown by the fact that he was the first man in England to make use of the X rays, when in 1895 he heard from a private source of the discovery of the Roentgen rays, he immediately set off for Germany to see for himself the photographic process by which bones could be shown through the body. It is significant of his love for children that the first X-ray taken in England was taken by him, with the help of Dr Thurstan Holland, to detect the presence of a bullet in a boy's wrist. Indeed, crippled and deformed children were his special care. Unlike Luther who advised the killing of all deformed infants his main work in life was curing or alleviating cripples, but specially was he concerned with crippled children.

Perhaps the chief benefit that this great surgeon conferred on the nation was due to his wonderful foresight, during the War in arranging for orthopaedic centres for the men who, he foresaw, would need all the help that surgery could give them when crippled and disabled by wounds and disease. These centres are now established all over England, and many surgeons are at work who were trained by Sir Robert Jones in the special knowledge needed.

The history of the first open-air hospitals for children is of great interest, and the book is well worth reading for that alone (p 114). There are many delightful stories in the volume, some gay, some sad, some mischievous, and all delectable. The marvellous capacity for hard work, the joy of living, the enjoyment of sport, the genius for friendship, and the fascination which Sir Robert exercised over our American colleagues are fully described, and though to those who did not know him the tale may seem incredible to those who did it rings true in every line. This account of a life spent in helping others should arouse enthusiasm in every heart.

The book is well produced, and contains 10 illustrations.

Intracranial Tumours By HARVEY CUSHING, Professor of Surgery, Harvard Medical School Super royal 8vo Pp 150 + vii, with 111 illustrations 1932 London Bailliere, Tindall & Cox 26s net

THIS volume really consists of a clinico-pathological elaboration of Professor Cushing's series of 2000 verified cases of cerebral tumour. Despite the title of the monograph, it can in no way be regarded as a representative account of intracranial new growths. This is shown by the author's references, which do not include any work carried out by individuals who have not studied at his clinic. The writer's experience is divided into three groups: at Johns Hopkins Hospital, from 1901-12, a Peter Bent Brigham series to 1922, and a third interval from 1922-31. The case and operative mortalities over the period 1901-31 have been compared with those in the three years 1928-31. Thus, over the total period the case mortality has been 20.4 per cent as compared with 13.3 per cent over the period 1928-31, and the operative mortality 13.9 as compared with 9.8 per cent. Operative fatalities comprise all cases of death in hospital following an operation, from any cause whatever, no

matter how long the interval. The average hospital sojourn of the last 100 cases with surgically verified tumours has been thirty-nine days, the author, however, does not specify the average stay in hospital after the actual operation.

Professor Cushing attributes the progress of intracranial surgery over the thirty years to three main factors: the development of a highly specialized surgical technique, more precise localizing diagnoses, and a better understanding of the life history of tumours of different types based on their histogenesis. In regard to the details of improvement in technique, the writer emphasizes seven features: (1) The generally accepted methods of decompression to relieve tension, (2) Such irreproachable wound healing that secondary infections are practically unknown, (3) The separate closure of the galea by buried fine black-silk sutures, which has made the once dreaded fungus cerebri almost forgotten, (4) In place of ether inhalation, the introduction by de Martel of local anaesthesia, now supplemented when necessary by the rectal administration of tribromethanol, (5) The more precise tumour localization which in obscure cases Dandy's ventriculography permits us to make, (6) The use of a motor-driven suction apparatus as an indispensable adjunct to every operation, and (7) The successive improvements in methods of hæmostasis which since 1927 have been most advantageously supplemented by the introduction of electro-surgical devices.

Professor Cushing himself realizes that mere improvement in operative statistics tells us comparatively little, and that what would be more desirable is an account of the remote history, the survival periods, and the working capacity of patients after extirpation of the tumour. As the writer admits, the ultimate case mortality of malignant cerebral gliomata is actually in the region of 100 per cent.

Professor Cushing likens his technical results to prowess at golf: "Games are scarcely worth playing unless one keeps a score. In golf, for example, it is only by the score that a player can tell whether his game is improving or falling off, and only when his competitors keep score by the same standards can it be told by comparison of figures whose game is the poorer." On the same analogy we may regard the writer's score as bogey.

The Adrenal Cortex: a Surgical and Pathological Study By L. R. BROSTER, O.B.E., M.A., D.M., M.Ch. (Oxon.), F.R.C.S., Surgeon to Charing Cross Hospital, and H. W. C. VINES, M.A., M.D. (Camb.), Pathologist, Charing Cross Hospital Institute of Pathology. Demy 8vo. Pp. 94 + vi, with 4 illustrations. 1933. London: H. K. Lewis & Co. Ltd. 6s. net.

It has long been recognized that some cases of virilism in women are associated with tumours of the adrenal gland; it is probable that their association is that of cause and effect, for removal of the adrenal neoplasm has been promptly followed by the disappearance of the virilism; moreover, in one case at least, the recurrence of the malignant growth was accompanied by the renewed onset of the signs of virilism.

The authors of this monograph start on the assumption that if a female shows the signs of virilism, there must be changes in the adrenal gland, and they show good reason for suggesting that this may be due, not to a neoplasm, but to a hypertrophy of the gland. As such a hypertrophy is unlikely to be obvious on clinical examination, they have opened the abdomen and palpated the adrenal on each side; if one was obviously larger than the other, they have at a second operation removed that one. The results of their operations are recorded in this book, and they are striking though not dramatic. Perhaps one could not expect the extraordinary return to normal that can occur after the removal of an adrenal tumour; there is always the possibility in their cases that both adrenals were hypertrophied, though not equally, or, after the removal of one, the other may take on the morbid change.

The last part of the book is occupied with a discussion of a material found in these adrenals which stains a vivid red with ponceau fuchsin; various reasons are given for considering this an indication of an over-production of a specific secretion.

In the excellent description of the various changes comprised under the heading of virilism we note with surprise one omission, whilst discussing the loss of hair on the head no mention is made of 'apical baldness', though we believe that this is a privilege reserved for the male; in one case on record the diagnosis of an adrenal neoplasm was first suggested by the presence of this limited alopecia.

The book is well written and must arouse great interest amongst surgeons, physicians, and pathologists.

The Thyroid Gland Its Chemistry and Physiology By CHARLES ROBERT HARINGTON, M A, Ph D, F R S, Professor of Pathological Chemistry in the University of London Large 8vo Pp 222 + xiv Illustrated 1933 London Oxford University Press 15s net

THE field of thyroid physiology is now so vast that Professor Harington is to be congratulated on the excellent and well-balanced manner in which he has treated the subject. The main theme of the book is to set out concisely yet completely the earlier work on the chemistry of the thyroid gland which led up to his own brilliant researches, and in more detail his own work and that of his collaborators on the chemical constitution and synthesis of thyroxine. On this there is no one better qualified to write, and even for this alone the book is to be thoroughly recommended.

To illustrate the biochemical relationships of the thyroid to the organism as a whole, he has given a reasoned account of the essential physiology and pathology of the gland. The importance of iodine deficiency, whether it be absolute or relative, in the etiology of simple goitre is justifiably emphasized, to bring forward arguments against the etiological significance of iodine is undoubtedly to lack a clear conception of the fundamentals of the subject. Professor Harington in his discussion on Graves' disease points out what pathologists are slowly beginning to realize, that the morphological changes and the biochemical reactions of the thyroid in this condition in no way differ from those of simple goitre, and shares the view of most competent observers that the cause of hyperfunction must be sought outside the gland itself. He forms the logical opinion that there is no good reason for considering the so-called "adenomatous goitre with hyperthyroidism" or "toxic adenoma" and Graves' disease as separate entities. In criticizing the dysthyroidism theory, however, his argument that the thyroid glands from cases of Graves' disease are inactive when administered to animals is unconvincing, for in the same way as we know that the gland pours out its active secretion at an increased rate so that none is retained, so the hypothetical "toxic" secretion would be poured out and not be retained. Moreover, many clinicians would not agree with his statement that the beneficial effect of iodine is but of short duration. The argument, however, which Professor Harington brings forward, that no derivative of thyroxine containing less than the full complement of iodine exhibits any toxic properties whatever, definitely refutes the hypothesis.

Professor Harington's book should be read by all those who are interested in the thyroid gland. There is a good bibliography.

The Study of Anatomy written for the Medical Student By S E WHITNALL, M A, M D, B Ch (Oxon), M R C S, L R C P, F R S (Canada), Robert Reford Professor of Anatomy, McGill University, Canada. Second edition. Crown 8vo Pp 93 + viii 1933 London Edward Arnold & Co 4s net

PROFESSOR WHITNALL, the author of this book of wisdom, is distinguished both as a writer and as an anatomist. He has generously put his reading and experience at the service of the medical student. If the student were to accept the advice offered and practise the precepts of the author, then he would be wise in his adolescence instead of only in his old age.

The author opens with a proper definition of anatomy and at once proceeds to make clear what the subject is. His insistence that topographers, mappers, sketchers, and markers of the body shall consider what the structures are for and how they work is welcome. An eloquent passage on the skin illustrates the author's attitude, and we quote its final sentences: "It [the skin] is indeed the finest fighting tissue. But it does surprisingly more than serve as a protective covering against such manifold influences, for it will also act as a regulator of temperature, an excretory organ, and the largest and most versatile of our sense organs."

The second chapter of the book is concerned with practical methods of study—sharpening scalpels, describing directly the dissected structure, drawing it, looking at other dissections, seeing the same region from some other approach or in cross-section, etc.

The author continues to act as Nestor in chapters on books, lectures, and examinations, to rub in common sense and plead for reflection and thinking as substitutes for memorizing—a drudgery that seems worse in medicine than in any other study. Point is given to common-sense remarks by a skill in apt quotation: "It is an idle and pernicious habit to ask for information on any question before bringing one's own judgment to bear on it" (Bridges).

The last chapter opens rather depressingly, for it begins with what one ought to read - then out come the usual first-past-the-post best hundred classics. Then the author, having put his penny in the cultural plate, proceeds in the raciest manner to string together the titles of his real bedside friends - *King Solomon's Mines*, *Treasure Island*, *Robbery under Arms*, and all the others.

Operating Room Procedure for Nurses and Internes By HENRY C. FALK, M.D., F.A.C.S., Clinical Professor of Gynecology, New York University, and Bellevue Hospital Medical College, etc. With Foreword by EUGENE H. POOL, M.D., New York. Second edition. Large 8vo. Pp. 413 + xxxii, with 328 illustrations. 1934. New York and London: G. P. Putnam & Sons. \$3.

THIS is certainly one of the most practical books on the subject which we have yet seen. It begins with a description of the principles and details of the operating room and then discusses the duties assigned to the various grades of nursing sisters, the design and making of the dress, etc., to be provided for surgeons and nurses, the provision of the operating theatre stores, and the best method of keeping them. The chapter on hand preparation is short, but the methods described are far more complicated than those used in this country. In order to preserve silence in the theatre, a manual signal system has been invented (which we confess we find it difficult to take seriously), thus by holding up the hand with one or more fingers turned in various directions the nurse is instructed to fetch some particular instrument or dressing. The larger second portion of the book consists of a description of various operations, chiefly those concerning the abdomen, pelvis, and female genital organs. The clearness of the line drawings is a commendable feature of the book.

La Sténose hypertrophique du Pylore chez le Nourisson By J. POUCEL, Chirurgien des Hôpitaux de Marseille. 7½ x 5 in. Pp. 108 + viii, with 16 illustrations and 8 plates. 1934. Paris: Masson et Cie. Fr. 20.

THIS little manual gives a very complete study of the clinical and pathological aspects of the disease. In stressing the necessity for early diagnosis followed by surgical intervention the author reminds us that Fredet practised and published his account of the modern operation some years before Rammstedt, whose name is commonly associated with the procedure. In diagnosis the author favours repeated short radiographic examinations, which he regards as harmless and essential. In this he would appear to differ from many English paediatricians who reserve the X-rays for the occasional doubtful case rather than for routine use, basing this practice on their experience that diagnosis in skilled hands is extraordinarily accurate, whilst the barium meal is not altogether harmless. We agree with the author that many lives are still being lost by a failure to recognize the condition in its earlier stages when its surgical treatment is safe and the results of operative intervention are most satisfactory.

The Enlarged Prostate and Prostatic Obstruction By KENNETH M. WALKER, F.R.C.S., M.A., M.B., B.C., Lecturer in Venereal Diseases, St. Bartholomew's Hospital, etc. Second edition. Demy 8vo. Pp. 223 + xiv, with 63 illustrations. 1933. London: Oxford University Press. 14s. net.

IN his preface to this edition the author explains the alteration of the title of this book (from *The Enlarged Prostate* to *The Enlarged Prostate and Prostatic Obstruction*) by the wider recognition of the part played by minor changes in the neck of the bladder and by the improvement in the instruments used for per-urethral resection; he points out that he is dealing with prostatic obstruction rather than with prostatic enlargement.

The reader will find an excellent account of the anatomy and function of the prostate, with some interesting remarks on the role played by the trigonal muscle in the act of micturition, and, in the third chapter, a very complete and well-balanced summary of the various theories on the pathology of prostatic obstruction.

We naturally look to the chapter on per-urethral prostatectomy as being of most interest to the modern surgeon; there we find a clear account of the operative technique and of the special instruments that have to be employed. Of even more importance at the present time are the author's remarks on the indications for this operation; he considers that the

key to success lies in the careful selection of cases and that the per-urethral approach is not to be looked on as a substitute for prostatectomy. He would employ this method for cases in which the intravesical portion of the prostate is affected whilst the total volume, as estimated by bimanual examination, is but little increased, and again for cases in which total prostatectomy is contra-indicated because the patient's general condition is too poor or when he is suffering from cardiac, pulmonary, or kidney lesions. In these conditions the author considers it an excellent alternative to catheter life or a permanent suprapubic drainage, and the fact that the obstruction may recur is not the drawback one might suppose, for the inconvenience is so slight that a patient will return for further treatment.

Not the least valuable part of this book are the references at the end of each chapter, they are well chosen and not too numerous.

Trattato di Patologia chirurgica generale e speciale By Professor OTTORINO UFFREDUZZI (Turin) Large 8vo Vol II Pp 1199 + vii with 25 plates and 448 illustrations Turin Unione Tipografico-editrice Torinese 1934 L 145

IN reviewing the first volume of this admirable work (BRIT JOUR SURG, 1933, xxi, 165) it was remarked that no acknowledgement was made of the plates taken from this journal's ATLAS OF PATHOLOGICAL ANATOMY. The omission has now been made good.

That one man should be able to cover the whole field of general surgery to-day is a notable tribute to his energy, his memory, and his catholicity of interest. That he should lend to twelve hundred pages an air of all having been written to-day is even more notable. This volume deals with the capillaries, veins, and lymphatics, the central and peripheral nervous system, the skin and its appendages, the respiratory and digestive systems, and the urogenital system. The book differs from, and is superior to, a great many large continental text-books, in that though a vast deal of current literature is traversed, it is all presented through the medium of the author's own mind, illustrates his practice, and here and there discloses his little prejudices. In every instance the general discussion of the physiology and pathology of a given system offers the student a reasoned and reasonable basis for diagnosis and treatment.

Only one or two points can be noticed. There is an account of successful experiments on the regeneration of the sympathetic after division, which is used as an argument in favour of ganglionectomy. In dealing with tumours of the lung, the accounts of lobectomy hardly reflect present-day success. The parathyroids have a good deal of consideration, but there are more specific results than are here indicated. In speaking of the physiology of the stomach the work of Bayliss and Starling, though perhaps implied, is hardly given due weight. Without, however, reading every word of the text attentively, it would not be fair to allege neglect of any piece of recent work, since again and again a casual sentence occurring outside the general discussions indicates a familiarity with contributions to the literature not specifically named amongst the ample acknowledgements.

An achievement of which the author and his University may be proud, the book deserves translation into English. It would be a colossal task, probably it will in fact be transcribed into American, and thus students here will have the benefit.

The 1932 Year Book of Radiology Edited by CHARLES A. WATERS, M.D., Associate in Roentgenology, Johns Hopkins University, etc (Diagnosis), and IRA I. KAPLAN, B.Sc., M.D., Director, Division of Cancer, Department of Hospitals, New York, etc (Therapeutics). Pp 750, with 495 illustrations. Chicago The Year Book Publishers, Inc \$6.00

THIS book contains abstracts of the most important articles which have appeared in the radiological journals of Europe and America during the past year. The abstracts are concise and clear, and a reference to the original article is given with each. The illustrations are numerous and maintain a high standard. There is an index both of subjects and authors at the end, so the book is ideal for reference purposes, while in many cases the abstracts are so clear that it will not be necessary to refer to the original, especially as these may be in German, while the abstracts are all in English. The book incorporates and summarizes all the recent advances in radiology and radium therapy during the past year, so that it should have a wide appeal to physicians and surgeons.

United States Army X-ray Manual Authorized by the Surgeon-General of the Army
 Second edition, rewritten and edited by Lieut -Colonel H C PILLSBURY, M D, U S A
 Crown 8vo Pp 482 + xvii, with 228 illustrations 1932 London H K Lewis
 & Co Ltd 25s net

WHEN the first edition of this work was published, it was found to be not only an excellent guide to war radiology, but also a very good elementary work on radiography in general. In revising the work the present writer has had the latter object more in mind than the former. The chapter on localization has been cut down from ninety-three pages to twenty-nine, and much that was useful has been omitted. For instance, the method of parallex which was extensively used in our Casualty Clearing Stations in the world war, as one of the quickest and most accurate, is not described. Surely the localization of foreign bodies must be taught to an army surgeon as long as rifles, machine-guns, shells, and bombs are issued to the rest of the army. When the use of these weapons discontinues, the need for an army X-ray manual will cease.

The war portion of the book has suffered little revision. For instance, the field unit described and illustrated is apparently the same as in the previous edition and is that used in the Great War fifteen years before. We civilians who worked in the British Army in the war found our commanders terribly conservative, but we trust they will in the next war be a little more abreast of the times than this. The need for an immediate and urgent report in cases of gas infection is not mentioned—a most important thing in a Base Hospital or Evacuating Station.

In the present edition an attempt has been made to separate technique into a distinct section, but the technique of some sections still remains in the text, e.g., in the chapter on sinuses. One of the features of the first edition was the excellent description and photographs of the various techniques employed in different parts of the body. These have now practically disappeared. Many illustrations have been added to the various sections, and some of them bear evidence of considerable revision, notably that on the thoracic viscera. Some sections are hardly brought up to date. We are glad to see that the chapter on therapy has been omitted in the original edition it was very bad.

Taking the work as a whole, therefore, it is to be regarded less as an army X-ray manual than as an elementary work on general radiography. Much that was valuable in the first edition has disappeared, and, though the work has been brought up to date, it is doubtful if the revision has proceeded along the best lines for the instruction of an army radiologist.

Radiologic Maxims By HAROLD SWANBERG, B Sc, M D, F A C P, Editor of *The Radiological Review*. With a Foreword by HENRY SCHMITZ, A M, M D, LL D, F A C P, F A C S. Large 8vo Pp 127 1932 Quincy, Ill. Radiological Review Publishing Co. \$1 50 net

THIS little book is made up of maxims on radiology and radium therapy, with short abstracts from the literature—or what one might call “famous sayings of the great.” Some of the maxims are good and some indifferent, while many are platitudes, such as (p. 39), “Roentgen examinations are indispensable to the urologist.” Generalizations are made which by themselves are not entirely true, such as (p. 102), “The therapeutic effects of radium and radon are the same.” It is difficult to see who is going to be instructed by these. Some of the statements are of interest as illustrating the views of the author or authorities quoted—for example (p. 36), “Duodenal ulcer is about ten times as frequent as gastric ulcer”, but even here the facts convey little unless amplified.

Radium Production, General Properties, Therapeutic Applications, Apparatus
 Demy 8vo Pp 346, illustrated 1933 London Chemical Service Co Ltd, Sunic
 House, Kingsway, W C 2

THIS is not a book written specially for medical men, though there are many points in it which are useful for doctors. It has been produced for the purpose of dealing briefly with the fundamental principles of radium therapy and of the precautions to be observed by the radium therapist. There are many points in it which will be found useful to those entering upon the study of radium therapy.

Radium and Cancer By H S SOUTTAR, CBE, MD, MCh (Oxon), FRCS, Surgeon to the London Hospital, etc, Pocket Monographs on Practical Medicine F^{cap} 8vo Pp 64 + viii, with diagrams 1932 London John Bale, Sons & Danielsson, Ltd 2s 6d net

THIS is a little book of sixty-four pages only and costing 2s 6d, yet it includes some of the most important facts relating to the treatment of malignant disease by radium. To anybody who is commencing the study of radium treatment of cancer this book will repay perusal. It describes quite briefly the physics of radium, the apparatus and means of application, the sensitivity of the tissues to radium, and details of the application of radium in various parts of the body. In addition it sets out in a final chapter a warning note against the accidents which too often have followed its indiscriminate use.

A Short History of Some Common Diseases By Divers Authors Edited by W R BETT Demy 8vo Pp 211 London Humphrey Milford (Oxford University Press) 10s 6d net

THE plan of this book is indicated by the title, although this gives no clue to the amount of information and interest which have been crowded into its pages.

It may be at once said that the dedication hides yet another illusion about the editor which may only be known to few, for it is a fact that Mr W R Bett who has assembled this remarkable collection of essays is not the old and satiated person that the dedication suggests, but one who ought to have many years of usefulness during which, let us hope, he will still further encourage the study of medical history, not as a detached subject but as a prelude to the knowledge and understanding of disease.

This little volume contains no fewer than sixteen articles, each one complete in itself, and dealing with most of the subjects under which morbid conditions are usually considered, starting with Acute Infectious Disease and finishing with Malingering. It includes subjects like Venereal Disease, Rickets, Endocrine Disorders, Arthritis, Tonsils and Adenoids and Malignant Disease, as well as others.

The names of the authors are a guarantee of the completeness with which the subjects are dealt. Sir Humphry Rolleston, Sir John Broadbent, Sir D'Arcy Power, E M Brockbank, and John D Comrie are all sufficiently well known as medical historians to command respect. Some of the chapters by less well known historians make delightful reading also, and the story of Tuberculosis by Professor John Fraser of Edinburgh, and that of Appendicitis by W R Bett, are especially worthy of mention.

In several of the articles there are quotations from older works and some of them give lists of references, but the way in which nearly every article unmasks the personality of its author makes the book the more delightful and valuable. How nice it would be if some future edition could be embellished by a few portraits!

This work is not only likely to attract the serious student, but will serve as an introduction to the history of the Healing Art, and it might well be expected that those preparing themselves for the higher reaches of the profession should be acquainted with its pages.

There are useful indexes both of subjects and names.

Lessons on the Surgical Diseases of Childhood By WILLIAM RANKIN, MB, ChB Royal 8vo Pp 190 Illustrated 1934 Glasgow Alex Macdougall 21s net

DR RANKIN tells us in his preface that the book is a summary of the lecture and demonstration notes which he gives to first-year students. He states that he intends it to be no more than a groundwork of introduction to the subject, and this is a correct description, for, while he covers a wide field in a superficial review, there is insufficient detail to make the book of value to the inquiring reader. For example, Chapter XI, which purports to deal with the common abdominal conditions of childhood, is condensed within a space of some thirteen pages including illustrations. It is obvious, therefore, that the volume is something in the nature of a catalogue of the surgical affections of childhood, elaborated at certain points in relation to detail which makes a special appeal to the author.

The volume is divided into twenty-six sections and of these the best is undoubtedly the first, dealing with hare-lip and other developmental lesions of the face. It is obvious

that this is a section in which Dr Rankin is particularly interested, and if the other sections had been brought to a similar standard the result would have afforded more interesting and satisfying reading than is the case under existing conditions. There are many points open to criticism, but these are too numerous to call for individual notice, it may, however, be permitted to refer to one or two. It is probably unwise to suggest that "only very rarely should the premaxilla be removed entirely." This would seem to presuppose that an incomplete removal may be adopted with relative frequency, but would it not be more correct to recommend that under no circumstances should the premaxilla be removed? This recommendation has the entire support of modern opinion. Again, in describing the operative treatment of empyema the statement is made, "the removal of two inches of the second rib beneath the angle of the scapula when the arm is laid against the chest wall is preferred to removal of a piece of rib in the mid-axillary line." It is probable that this statement is the result of imperfect wording, but as it reads it would seem to suggest that a portion of the second rib should be removed. Presumably the author intends to recommend removal of a rib at a lower level than the scapular angle, i.e., the 9th or 10th rib, but as it stands the statement is apt to create an entirely wrong impression in the student mind. We question, too, whether it is judicious to state that "in empyema the old operation of free drainage through a fairly large drainage tube has not been replaced by any of the more modern methods." It is a pity that no attempt has been made to distinguish between the kind of treatment adopted in the pneumococcal and in the streptococcal types of the disease.

Dr Rankin, in speaking of the operation for mastoid infection, says that once one is familiar with the small area involved and the danger spots, the operation is easy, and can be performed with a small Volkmann spoon. This is surely an optimistic view to adopt, and we question if it would have the approval and the blessing of those who are interested in operations of this class.

We are in strong disagreement with some of the author's views on embryology and comparative anatomy. It is surely disrespectful, to say the least of it, to speak of the teratoma as a 'blasted ovum', and there is no evidence that webbed fingers constitute a throw-back to the flipper limb of an amphibian ancestor. We disagree, too, with the statement that the development of the spinal cord is an antenatal operation necessitating the making and the healing of a huge wound. This statement may have certain attractions in the way of descriptive writing, but it is scarcely an accurate presentation of the facts.

The volume is profusely illustrated, though a certain number of the photographs are inserted without descriptive legends. The value of the book would be increased by the addition of an index. The printing of the text and the presentation of the book from the publisher's point of view leave nothing to be desired.

Bone Growth in Health and Disease By H. A. HARRIS, D.Sc., M.B., B.S. (Lond.), M.R.C.S., M.R.C.P., Professor of Clinical Anatomy, University College and University College Hospital, London, etc. Crown 4to. Pp. 248 + xv.ii, with 201 illustrations. 1933. London: Humphrey Milford (Oxford University Press). 34s. net.

IN this interesting and discursive monograph, Professor Harris elaborates views on bone growth which he has already published elsewhere.

The first part deals with the lines of arrested growth in the long bones of children which form during a period of starvation, infection, or disease.

The second part is concerned with bone growth in deficiency diseases. The problem is approached from two aspects: (1) The general principles of ossification in the skeleton, and (2) A detailed consideration of the changes seen in proliferating cartilage, calcifying cartilage, and bone in terms of histological, radiographic, and morphological criteria. Emphasis is laid on the differences of nutrition in these three processes, and it is suggested that each is controlled or influenced by a different vitamin, and by other substances of a somewhat speculative nature.

The origin, function, and fate of the osteoblast, and of the osteoclast, are still debatable problems. The author believes that the osteoblast is an altered cartilage cell possessing specific bone-forming properties, but presents no new or convincing evidence in support of this traditional view. It would have been instructive if he had discussed in critical fashion the views of Leriche and Policard, and Greig, that bone formation is merely a metaplasia of

fibrous connective tissue in which calcium and phosphorus are deposited, and that the osteoblast is simply an altered fibroblast with little, if any, bone-forming property

Part III is devoted to a study of the significance of the fundamental processes of growth and repair in skeletal disease. This section is stimulating and provocative. From observations of the cells of normal human cartilage it is claimed that the dividing cells are arranged in a 'mitotic annulus' at the end of a bone, and from this limited area growth occurs. The author believes that synovial fluid is produced by the disruption of the cells of articular cartilage and is absorbed by the synovial membrane.

Harris has carried the researches of Robinson and Fell on the ferment phosphatase to a further stage, and has demonstrated the gradual accumulation of glycogen in the cartilage cell as the cell ages, to disappear entirely when cartilage is replaced by bone. He suggests that the glycogen is hydrolysed to hexose phosphoric esters which under the action of phosphatase and the calcium of the circulating body fluids leads to the deposition of an insoluble phosphate of calcium in the matrix. How in the absence of a blood-supply calcium reaches the cartilage cell is a problem that requires further research, but it seems certain that the explanation of calcium deposition is a biochemical, and not a mechanical one.

Although this book is fragmentary in its presentation of the subject of bone growth, it makes attractive reading, and should give fresh stimulus to research on a problem which is still to be solved.

The Spread of Tumours in the Human Body By RUPERT A. WILLIS M.D., B.S., D.Sc. (Melbourne), Pathologist to the Alfred Hospital and to the Austin Hospital for Chronic Diseases, Melbourne. Demy 8vo. Pp. 540 + 1, with 103 illustrations. 1934. London: J. & A. Churchill. 25s. net.

It is unusual and most refreshing to find a pathologist, or for that matter any other medical author, attacking a subject from an original point of view and departing completely from the seissors-and-paste methods so common and so tedious in medical literature. Dr Willis believes that a close study of the modes of spread of tumours may help to teach us much of their nature and origin, and his book, the outcome of many years of careful study of his own autopsy material and of exhausting sifting of an immense volume of literature—his bibliography fills 65 pages and contains more than 2000 references—is an ample justification of his claims.

The book is divided into two parts. In Part I the general problem is discussed, and excellent chapters appear on the routes of dissemination, the reaction of stroma, and the comparative susceptibilities of different tissues to tumour growth, etc., while Part II deals with secondary neoplastic disease of individual organs and tissues.

The book is full of valuable information and carefully weighed criticism of existing theories, criticisms which are inspired by a rare soundness of judgement. It widely exceeds the limits suggested in the title, for it is a general exposition of tumour formation, and those who delight in theory will be disappointed, for the writer deals with facts and discusses theories mainly to explode them. Dr Willis apologizes in his preface for appearing iconoclastic, but his outspoken criticisms are one of the most attractive features of the book, and he has done a great service in exposing the fallacies of much teaching that has become traditional.

In a book which is in every respect so excellent it is difficult to select parts for special commendation. It will appeal to all lovers of truth, and to the morbid anatomist and surgeon alike its value cannot be exaggerated.

Spinal Anæsthesia: Technique and Clinical Application By GEORGE RUDOLPH VEHRIS, M.D., Salem, Oregon. Large 8vo. Pp. 269, with 81 illustrations. 1934. London: Henry Kimpton. 21s. net.

ANY reader unacquainted with the practical side of spinal anæsthesia reading this book might excusably make the deduction that the question of anæsthesia for any operation in surgery had been solved so perfectly that it would be scarcely worth while to make any further researches on this subject. From which it is clear that the author is an enthusiast for the method. He seeks to convince us that, by the adoption of his technique, the anæsthesia is absolutely controllable. The sequence of events is fixed by what he is pleased to call "unalterable laws of spinal anæsthesia." He starts out with the assumption that novocain introduced

into the spinal canal diffuses only by the influence of gravity. He relies upon fixation of the novocain locally to reduce its concentration to a value harmless by the time it has risen to the cervical region. He advocates keeping the patient exactly horizontal on his side for some minutes after injection to obtain this fixation before adopting the Trendelenburg position to cause upward spread. There is nothing new in his technique but this.

An interesting chapter is devoted to regional spinal anæsthesia. By this is meant sensory without motor block, which according to the author it is possible to obtain at will by reducing the dose of the drug, because a higher concentration is needed for motor than for sensory block.

There are a number of experiments described in which the drug was injected into the cisterna magna of dogs which prove that if the concentration is high enough death will follow. In view of Koster's statements it is well to have this fact which others have demonstrated insisted upon.

The book does not add very much to our knowledge of this interesting subject.

Verletzungen und Krankheiten der Kiefer By GEORGE PERTHES (Tubingen) and EDUARD BORCHERS (Aachen). Royal 8vo. Pp 623 + xiv, with 234 illustrations. 1932. Stuttgart. Ferdinand Enke. Paper covers, RM 75, bound, RM 78.

THE first edition of this book was published twenty-five years ago by Perthes. The second edition has been prepared by the pupil of Perthes—Eduard Borchers. The work comprises a complete treatise on affections of the jaws together with a very extensive bibliography. References are made to the English and French literature on the subject, and the arrangement of the book is effective. Deformities are dealt with fully, due regard being paid to congenital anomalies. Acquired deformities are discussed in acromegaly, cretinism, idiocy, thymus affections, and rickets. The treatment of fractures of the jaws is given fully, and the importance of the help of a dental surgeon is stressed. Dislocation of the mandible, diseases of the temporo-mandibular joint, and inflammation in all its varieties are exhaustively surveyed. The section devoted to neoplasms is particularly good. The question of the classification of odontomata is discussed, and a simple classification put forward. Regarding the treatment of sarcomata, we note the advisability of radical operation instead of spending time using radiological methods, which are not followed by any striking improvement. The author finds carcinoma more common than sarcoma affecting the jaws, and recommends radical removal where possible.

The book is well worth study. The print is clear and the illustrations, histological, naked-eye, and radiological, are good.

Gelenksteifen und Gelenkplastik By Professor Dr ERWIN PAYR (Leipzig). Vol I. 11 x 8 in. Pp 380 + xiv, with 240 illustrations. 1934. Berlin. Julius Springer. Paper covers, RM 120, bound RM 124 8.

IT is difficult to do justice to this magnificent monograph in the space at our disposal. For more than thirty years it has been in preparation. The author had at first planned a book dealing with the technique of plastic joint reconstruction, but he has been so greatly impressed with the need for laying a good foundation of biology, pathology, anatomy, and biochemistry that at present he has had to be content with the presentation of these aspects of the problem with which are associated a rich fund of clinical material and experimental evidence. A further volume is promised which will deal with the technique of operative procedures.

The opening chapters deal with the history of the subject, the development of joints, comparative anatomy, and composite structure of joints in animals and man. Then follows a description of the origin, nature, and causes of different types of ankylosis in constitutional diseases, infections, and injuries. The pathological anatomy and physiology of ankylosed joints are described in great detail and then the phenomena of the formation of false joints. After this natural formation of false joints there follows a discussion of the experimental attempts at joint regeneration. This section concludes with a most instructive summary of the application of the lessons of animal experiments to the human problem. The most significant point is that in animals the rapid regeneration of a joint largely depends upon its early active use and it is argued that this principle should be applied in human surgery.

The clinical aspects of ankylosis are analysed with great care. Kinetic factors, psychology, pain, and tonus are all discussed. The fibrous and bony types of ankylosis, the effect of

ankylosis on the rest of the body, and the compensatory changes which take place are considered in connection with each individual joint. A consideration of the various causative factors in ankylosis, congenital, inflammatory, infectious, constitutional, and endocrine disturbance, is followed by that of the technical methods of determining its nature and cause. This volume of the work concludes by a lengthy discussion of prognosis and the indications or contra-indications for plastic operations on ankylosed joints.

The work is of a thorough and monumental character, it is well printed and beautifully illustrated, and not only the many pupils, past and present, of its distinguished author, but the whole surgical world is richer for its appearance.

Synopsis of Surgery By ERNEST W. HEY GROVES, M.S., M.D., B.Sc. (Lond.), F.R.C.S., Consulting Surgeon to the Bristol General Hospital, etc. Tenth edition. Crown 8vo. Pp. 693 + viii, with 163 illustrations. 1933. Bristol: John Wright & Sons Ltd., 17s. 6d. net.

IN the latest edition of this well-known book the whole text has been carefully revised and brought up to date. The sections on the radium treatment of malignant disease, the surgery of the sympathetic nervous system, and the vaseline pack or Winnett-Orr method of treating septic bone conditions have been re-written, and a new chapter has been added giving an excellent outline of the principles underlying and the features of the more important amputations. The fact that this is the tenth edition of the book that has appeared since its original publication in 1908 is sufficient guarantee of its continued usefulness to the student and of its popularity.

Il Cancro del Retto By VITTORIO PETTINARI. Crown 4to. Pp. 225 + iv, with 121 illustrations. 1934. Bologna: Licinio Cappelli. L. 30.

THIS monograph, written by one of Professor Castiglioni's staff in the Institute of Surgical Pathology in the Royal University of Milan, is based on an extensive material and upon wide study of the literature.

It deals systematically with every aspect of the subject, and is fully illustrated, most of the pictures being adequate. It cannot be said that anything very new is incorporated, either in the pathogeny, diagnosis, or treatment, and for students there is rather too much record of numerous other surgeons' opinions, without clear indication of what the author himself thinks. He has, he says, frequently observed the initial stages of cancerization, and considers that differences are recognizable between the changes occurring when it commences in normal tubules and adenomata respectively. The symptomatology, clinical forms, and diagnosis present nothing exceptional, though it is noteworthy that the proliferative type seems more frequent in Italy than the ulcerative. Statistics are collected from all sources. The operative measures recommended do not commend themselves. Colostomy is used only when obstruction actually exists, and the lower end of the colon, after perineal operation, is abandoned in the depths of the wound, which is allowed to granulate under Carrel-Dakin dressings. There is a good bibliography.

Die Embolie By Dr. SIGURD FREY (Königsberg). Royal 8vo. Pp. 178, with 36 illustrations. 1933. Leipzig: Georg Thieme. RM. 12.

THIS is a monograph in German by Dr. Frey, the Privatdozent for Surgery in the University of Königsberg. It is a description of the varieties of emboli, their causes, and their effects.

Beiträge zur Mund und Kieferchirurgie By Prof. Dr. G. AXHAUSEN (Berlin). Part 2 of *Deutsche Zahnheilkunde*, edited by OTTO WALKHOFF (Berlin). Large 8vo. Pp. 117, with 128 illustrations. Leipzig: Georg Thieme. M. 12. 60.

A USEFUL contribution to the surgery of the mouth and jaws has been made by Axhausen. A work of this size is not intended to cover the whole field, the main aspects of the subject have been dealt with. Numerous clinical histories are given to illustrate various facts. The pictures are very good. The subject of plastic surgery of the face has been introduced. Profit will result from a perusal of this book.

Chirurgie de la Main Plaies, Infections, Chirurgie réparative By Dr MARC ISELIN
Ancien Interne des Hopitaux Royal 8vo Pp 339 + xii, with 111 illustrations 1933
Paris Masson et Cie Fr 55

THIS monograph deals with wounds, infections, and the reparative surgery of the hand In it the author has incorporated the work of Kanavel, Bunnell, and others From an extensive personal experience at the St Louis Hospital, Paris, he advocates some useful technical modifications, e g, in regard to skin incisions for drainage, the site for drainage of tendon-sheaths, and so on, but on the whole he conforms with modern surgical practice The book is an excellent work of reference

Kleine Chirurgie By HANS KURTZAHN (Königsberg 1 Pr) Second edition Large 8vo
Pp 462 + viii, with 167 illustrations 1932 Berlin and Vienna Urban and Schwarzenberg Paper covers, RM 13 50, bound, RM 15

WE are impressed with the practical value of this book The methods advocated are represented pictorially The chapter on bandaging we think might have been more comprehensive Anaesthesia in all its forms is discussed fully The various types of dental forceps are illustrated, and the methods of teeth extraction are given The book can be recommended as a short surgical work

Allgemeine und spezielle chirurgische Operationslehre By Dr MARTIN KIRSCHNER (Tubingen) Vol V/1 Die operative Beseitigung der Bauchbrüche (M KIRSCHNER) Die Eingriffe an den weiblichen Geschlechtsorganen (G A WAGNER) Royal 8vo
Pp 395 + v, with 304 illustrations 1933 Berlin Julius Springer Paper covers, RM 78, bound, RM 86

THIS is an operative surgery book dealing with hernia of various descriptions, and with gynaecological operations It is well illustrated with a large number of coloured plates, but has nothing particular to recommend it in preference to similar publications in the English language

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers]

The Bristol Medico-Chirurgical Journal Combined Index for Volumes 1-50 (1883-1933) Medium 8vo Pp 72 1934 Bristol J W Arrowsmith Ltd 3s

Anatomy of Animal Types for Students of Zoology By E A BRIGGS, D Sc, Assistant Professor of Zoology, University of Sydney Demy 8vo Pp 250 + xxii 1934 Sydney Angus & Robertson Ltd London Australian Book Co 10s 6d net.

Acute Intestinal Obstruction By MONROE A McIVER, M D, Surgeon-in-Chief, Mary Imogene Bassett Hospital Cooperstown, N Y 10¹ / 7 in Pp 430 + viii, with 62 illustrations 1934 New York Paul B Hoeber Inc \$7 50

The Clinical Management of Horseshoe Kidney By ROBERT GUTIERREZ, A B, M D, F A C S, Chief of Clinic of the Department of Urology, James Buchanan Brady Foundation of the New York Hospital, etc With a Foreword by Dr EDMOND PAPIN (Paris) 10¹ x 7¹ in Pp 143 + xiv, with 52 illustrations 1934 New York Paul B Hoeber Inc \$3 00

La Pratique chirurgicale illustrée By VICTOR PAUCHET Fasc XIX Super royal 8vo. Pp 295, with 249 illustrations 1934 Paris G Doin et Cie Fr 70

Benign Tumours in the Third Ventricle of the Brain Diagnosis and Treatment By WALTER E DANDY, Adjunct Professor of Surgery, the Johns Hopkins University 10 x 6¹ in Pp 171 + viii, with 120 illustrations 1934 London Baillière, Tindall & Cox 22s 6d

- Bulletin der Schweiz Vereinigung für Krebsbekämpfung** Herausgegeben unter Mitarbeit des Eidg Gesundheitsamtes February, 1934 1st year, Fasc 2 Large 8vo Pp 81-144, with 30 illustrations 1934 Bern Hans Huber No price given
- The Story of the Royal Infirmary, Sunderland** By WILLIAM ROBINSON, M S, M D (Dunelm), F R C S Large 8vo Pp 135 + vi, with 10 plates 1934 Sunderland Hills & Co (Sunderland) Ltd Cloth, 4s, paper covers, 3s
- Lord Lister the Discoverer of Antiseptic Surgery** By C J S THOMPSON, M B E, Hon Curator of the Historical Collection of the Museum of the Royal College of Surgeons of England Crown 8vo Pp 99 Illustrated 1934 London John Bale, Sons & Danielsson, Ltd 5s net
- Die parasagittalen Meningiome** By Dr H OLIVECRONA (Stockholm) 10½ / 7½ in Pp 144, with 145 illustrations 1934 Leipzig Georg Thieme Paper covers, M 24, bound, M 26
- Die pathologisch-anatomischen Grundlagen der Chirurgie des Rektumkarzinoms** By Priv.-Doz Dr HEINRICH WESTHUES (Erlangen) With a Foreword by Prof Dr SCHMIEDEN (Frankfurt a M) 11½ 8 in Pp 113 + xii, with 107 illustrations 1934 Leipzig Georg Thieme Paper covers, M 29 50, bound, M 32
- Narkose zu operativen Zwecken** By Dr HANS KILLIAN (Freiburg i Br) Super royal 8vo Pp 406 + viii, with 165 illustrations 1934 Berlin Julius Springer Paper covers, RM 24, bound, RM 26 80
- Die Wirbelgelenke** By Priv.-Doz Dr MAX LANGE (München) 10 6½ in Pp 121 + vi, with 59 illustrations 1934 Stuttgart Ferdinand Enke RM 7
- X-ray and Radium Injuries Prevention and Treatment** By HECTOR A COLWELL, M B, Ph D, M R C P, D P H, and SIDNEY RUSS, C B E, D Sc, F Inst P, the Barnato Joel Laboratories, Middlesex Hospital Demy 8vo Pp 212 + xii Illustrated 1934 London Oxford University Press 14s net
- The Management of Fractures, Dislocations, and Sprains** By JOHN ALBERT KEY, B S, M D, Clinical Professor of Orthopedic Surgery, Washington University School of Medicine, and H EARLE CONWELL, M D, F A C S, Orthopedic Surgeon for the Tennessee Coal, Iron and Railroad Company, Birmingham, Ala Imperial 8vo Pp 1164, with 1165 illustrations London Henry Kimpton 63s net
- The Origin of Cancer** By J P LOCKHART-MUMMERY, M A, M B, B C (Cantab), F R C S, Senior Surgeon, St Mark's Hospital, etc Large post 8vo Pp 150 + v, with 29 illustrations 1934 London J & A Churchill 10s 6d net
- Manipulative Treatment for the Medical Practitioner** By T MARLIN, M D, Ch B, D P H, R C P S (Eng), D M R E, Medical Officer in Charge of the Massage, Electrotherapeutic, and Light Departments, University College Hospital, etc Demy 8vo Pp 133 + vii, with 86 illustrations 1934 London Edward Arnold & Co 10s 6d net
- Surgical Anatomy and Physiology** By NORMAN C LAKE, M D, M S, D Sc (Lond), F R C S, Senior Surgeon, Charing Cross Hospital, etc, and C JENNINGS MARSHALL, M D, M S (Lond), F R C S, Surgeon, Charing Cross Hospital, etc Demy 8vo Pp 888 + v, with 238 illustrations 1934 London H K Lewis & Co Ltd 30s net
- Aids to Operative Surgery** By CECIL P G WAKELEY, D Sc (Lond), F R C S (Eng), F R S (Edin) Second edition 6½ x 4 in Pp 225 + viii, with 3 illustrations 1934 London Baillière, Tindall & Cox 3s 6d net
- Abscess of the Brain its Pathology, Diagnosis and Treatment** By E MILES ATKINSON, M B, B S (Lond), F R C S, Jacksonian Prize Essayist, R C S, 1926, etc Medium 8vo Pp 289 + x with 45 illustrations 1934 London Medical Publications Ltd 21s net
- St Bartholomew's Hospital Reports** Edited by LORD HORDER, R G CANTI, W SHAW, C F HARRIS, H H WOOLARD, R C ELMSLIE, W G BALL, G EVANS, and J P ROSS Vol LXVI Demy 8vo Pp 363 + xiv Illustrated 1933 London John Murray 21s net
- Diathermy in General Practice** By ERIC PAYTEN DARK, M C, M B, Ch M (Syd), late Radiographer, Royal Prince Alfred Hospital, Sydney With special chapters by F A MAGUIRE, C M G, D S O, M D, Ch M (Syd), F R C S, F R A C S, and GUY P O PRIOR, L R C P, M R C S Second edition Demy 8vo Pp 219 + xvi Illustrated 1935 Sydney Angus & Robertson Ltd 17s 6d net

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IPSISSIMA VERBA

BY SIR D'ARCY POWER, K B E, LONDON

III TWO PRE-HUNTERIAN OPERATIONS FOR ANEURYSM

JOHN HUNTER would sometimes have saved himself trouble had he been more conversant with the literature of his own profession. He took infinite pains to study the effects of arterial ligature before he tied the femoral in its continuity to cure an aneurysm at St George's Hospital in December, 1785. Richard Wiseman (1622-86) records two cases in which he tied the main artery more than a hundred years earlier. Both operations were undertaken in the ordinary course of his surgery, and both were successful. The first case was certainly done before 1672, for the details of it are printed in the rare first edition of his works*. He says —

“A Cooper living near Maidenhead in the County of Bucks, accidentally in letting blood was prickt in an Artery, the Arm swells and is pained, he puts himself into another Barber Chirurgions hands dwelling in Windsor, who by unfit applications, Relaxes the part, then supposing the soft Tumor he had made was a Suppuration of Matter, (though indeed it was the Arterial blood) he with his Incision knife or Lancet cuts into it, at which an impetuous flux of blood rushes out, to the quantity of four flaggons, he fills up the opening with Lint, and makes a hard bandage round it, which somewhat restrains the flux of blood, but the Arm swells, and threatens a Gangreen

“While this poor man lay thus afflicted, We, his Majesties and Royal Highness Chirurgeons attending the Court, did Visit him, and proposed to make an apparatus for the taking up this Artery, or in case of failing to take off his Arm by Amputation, We met the next morning, the Patient is taken out of bed and placed in a Chair towards the Light, one of his friends held him in the Chair. Mr Whittle stood behind his Arm, and held his Elbow with his left hand, and with the other hand was ready to help me in the operation, Mr L holds the lame hand

“In the first place I viewed the Arm, where finding no Gangreen according to the report of the Chirurgeon, but rather (as I thought) an Aneurisma, I made

* *A Treatise of Wounds* by Richard Wiseman, 8vo, London, 1672, p 43. Printed by R Norton for Richard Royston, Bookseller to His most Sacred Majesty

a bandage above the wound, to hinder the Influx of blood into it Upon this bandage Mr Pearce made the gripe, I then untied the Ligature from about the wound, and found that also free from Mortification, I put my finger into it, and finding the skin made thin by distention from the blood, I call'd for a Knife, which while I was searching in the wound, Mr Whittle took into his hand I prayed him to cut through the length of the hollowness It being done, I with my finger thrust out the Grumous blood and an Absces from the lower part of the Arm When I had cleared the parts more distant, which would have fallen in and blinded my Work, I then made way to the Artery, removing from about it the clotted blood, upon which it immediately burst out, I prayed Mr Whittle to hold his finger upon it, he did so, then I separated it from the parts about it, and passed a crooked Needle under it, and being ready to tie it, I desired Mr Whittle to hold off his finger, that I might be the more sure it was the Artery, upon the doing of which it spurted out I tyed it, but in tying the Thread broke This caused a murmuring in some of the by-standers, as that the Patient would die under our hands while I was endeavouring deligation, which by a new wounding of the parts would hasten Mortification and Death, I desired they would continue their places, and Mr Whittle his finger upon the Artery, that it bled not, he did so, in the while I made a strong Ligature of some twisted Threads, seared it, and put it into the eye of one of our common silver searching-Probes Gentlemen, said I, you shall see me pass this Probe under the Artery, and thereby you may be assured I shall wound no parts, I immediately did so and tyed the Artery, Mr Whittle took off his finger, it bled not, then Mr Pearce took off his hands, I then loosened the Bandage, which was all the time before under his hands, It bled no more

"I told them there was more to be done in order to the binding that Artery, but in consequence of the continual Fainting of the Patient, we would defer it to the next dressing then calling for dressings, they brought me what was designed for Amputation, which I refused, and ordered the common digestive *e Terebinth cum Vitell ovi*, which they fetcht I cut off the ends of the Ligature, and dipt some of the pledgits spread with the Liniment in Galen's powder, and applyed them next the Artery, and the rest of the pledgits dipt in *ol Ros* warm, and lightly fill'd up the wound, then Embrocated the Arm with the same oyl, and laid a *Diacalcitheos* Emplaster over the wound and that part of the Arm, and over the hand and Arm below which was Oedematous, a mixture of *Diacalcith Paracels &c* Then with bandage began at the hand and rouled up the wound, and taking a turn or two there, I rouled up to the Axilla This was the *Ligatura Expulsiva*, by which I proposed the thrusting back the Influx of humors, and to give strength to the infeebled Member, He being now again in his Bed, I placed his hand upon his breast in much ease to the great joy of the Patient

"At the next dressing, only Mr Whittle and the Countrey Chirurgeons were present with me, we opened the Member, found the tumor allay'd, the wound fresh and tollerably digested, we now passed another Ligature upon the Artery above the first, and in pulling the first to cut it off between the Ligatures, it broke, which was as well We drest it up with the same digestive, only leaving out the Powders, Embrocated and applied our Emplaster, rouled up the parts as before

"The next time Mr Whittle and I undrest the member, and found the wound in a very good condition, with assured hopes of curing it in a few weeks, the Lips of the wound being more digested and contracted, we drest it up with *Mund*

Paracels And now being Impatiently expected at London, with my diseased Patients, I return, leaving it to Mr Whittle, who drest it for the space of 10 dayes, it healing up without any return of bloud, In his absence Mr L dressed the Patient, but whether in wiping off the knot or what else, it burst out again, Sergeant Knight came in, dressed it with *Calcanth ust* and from that time it bled no more

“ The Ancient way of tying this Artery is, as I have already said, by tying it in two distinct places, and cutting it off between, but here the Patient often fainting interrupted us in that work, and the breaking between saved us the labour ”

The case is reported again in Wiseman's *Several Chyrurgical Treatises* (p 356), published in 1676, but in preparing it for the press he modified the account

Of the actors in the scene, John Knight took his B A from Caius College, Cambridge, in 1622, was surgeon to King James I, and was Serjeant Surgeon to Charles II in 1661 Mr Pepys records that he “ dined with Mr Knight, chief chyrurgeon to the King, and his wife ” on New Year's Eve 1662 “ We were pretty merry, he being excellent Company ” Richard Wiseman was appointed Serjeant Surgeon in 1672 and was Master of the United Company of Barbers and Surgeons in 1665 James Pearce was surgeon to the King, Charles II, and to the Duke of York, afterwards King James II He was Master of the Barber Surgeons Company in 1675 Mr L may possibly have been Lawrence Loe, the King's Barber, who was Master of the Barber Surgeons Company in 1667 It is more likely, however, that he was the local practitioner whose name can no longer be recovered

(*To be continued*)

FRACTURE OF THE FEMUR WITH LUXATION OF THE IPSILATERAL HIP *

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INTRODUCTION

IT has been our fortune to treat the only two cases of this rare association of injuries that were recorded in an aggregate of 9945 fractures and luxations at Kasr el Aini Hospital during the past eight years (1925-32). In this period the patients admitted to the wards numbered 151,663. These two cases, therefore, of femoral fracture with hip-luxation form an incidence among the total admissions during these eight years of 0.0013 per cent, a rate of 13 per 1,000,000. Their incidence in the aggregate of all the luxations and fractures treated at Kasr el Aini Hospital in the same period is 1 case in about 5000, or 0.02 per cent.

Future studies of larger series will exactly define the rarity of hip-luxation with femoral fracture, meanwhile the Kasr el Aini figures show that the combined injury is rare.

An account of our two cases will serve to preface a study of the different varieties of this fracture and luxation, which is based on all the records that we could find in the small and scattered literature of the subject. We shall first set aside the fractures caused by attempts to reduce luxations of the hip, and shall postpone the consideration of these surgical accidents till we have dealt, in *Part I*, with the 42 cases in which the luxation and fracture have formed successive events of an immediate traumatic sequence. The first in this sequence is probably always the luxation, but we have decided to classify the varieties of the combined injury in terms of the fractures. This classification will be more readily accepted than one according to hip-luxation, for which definitive terms have not yet been fixed. Further, it is the break in the femoral lever that dominates the question of treatment. The cases, therefore, which we believe to be authentic will be grouped under four headings according to whether the femoral fracture affected (1) the head, (2) the neck, (3) the shaft, or (4) some other portion of the femur. Cases which at any time have passed muster in previous accounts, but where the diagnosis of the combined injury has appeared questionable, will be summarized in footnotes.

THE AUTHORS' TWO CASES

Case 1—Fracture of the femoral shaft with hip-luxation—

K. A., a strong, healthy, woman, aged 64 (Hospital No. 15055, year 1928), was admitted to Kasr el Aini Hospital on Nov. 19, 1928, with a fracture of the right femur in the upper

* From the Surgical Unit, Kasr el Aini Hospital, Cairo

third, 6 in distal to the top of the great trochanter, and a luxation of the head of the right femur on the dorsum ili, both conditions being found by X-ray before she was referred to us (Fig 144). The total shortening due to the two injuries was about 4 in. The proximal fragment was markedly flexed, while the distal was drawn up behind it.

One unsuccessful attempt to reduce the luxation by 'closed' methods had been made before she was admitted to our Surgical Unit, and after admission a second attempt was equally unsuccessful. By this time three weeks had elapsed since the original injury, and we believed that nothing but an 'open' operation could give us sufficient control of the short upper fragment to effect reduction of the hip.

OPERATION — After preparing the patient's rather rough dry skin for four days with a 1 per cent aqueous solution of mercurochrome, we made the ordinary Smith-Petersen incision for exposing the hip-joint, continuing it well down into the middle of the thigh, after the method described by one of us, to expose the femoral shaft. There was a negligible loss of blood and the condition of this hale and energetic patient was admirable throughout the operation. The incision gave an ample exposure of the joint and of the site of fracture, but it was impossible, even with lion-forceps, to apply sufficient traction on the upper fragment to reduce the head. To get better control we bored through the upper fragment just below the great trochanter and passing a 2-ft length of $\frac{1}{8}$ -in iron wire through the hole we bent it into a ring, and with padding made it comfortable for manual traction. After pulling for about one minute, the head, pushed also from behind, suddenly slipped into place, and so silently that we at first were unaware of its reduction, the fact of which, however, we soon verified in the wide wound. After removal of the wire ring, and partial reduction of the fracture by manipulation, the muscular planes were closed with interrupted sutures of unchromicized gut, skin-closure was effected entirely with Michel clips. Strapping was then applied to the leg and lower part of the thigh, and the limb was extended by traction in full abduction in a straight Thomas splint. The wound healed perfectly and the patient's convalescence was uneventful, though her energy more than once threatened to compromise the undisturbed union of the fracture.

An X-ray taken a fortnight after the operation confirmed the presence of the femoral head in the acetabulum. The reduction of the fracture, however, was only approximate, but was nevertheless sufficiently good, and we did not again disturb the patient. (See Fig 145, from an X-ray three months after operation.)

AFTER-TREATMENT — Massage was begun in the third week after operation, and on Dec 29, 1928, forty-seven days after operation, a plaster spica was applied. This was changed twenty-eight days later, and on

March 4, 1929 (113 days after operation), she walked with a caliper, in which she was discharged.

END-RESULT — Two years later, in May, 1931, with the greatest difficulty—for this nomadic person had changed her address fourteen times—the patient was brought back to Kasr el Aini Hospital for examination, largely through the efforts and courtesy of Ahmed Moukhtar Pasha Hegazi. Her condition is well seen in the accompanying photographs (Figs 146-148).



FIG 144—Case 1. The fracture of the femoral shaft and the hip-luxation.



FIG 145—Case 1. The hip luxation reduced by operation. The shaft-fracture has united.

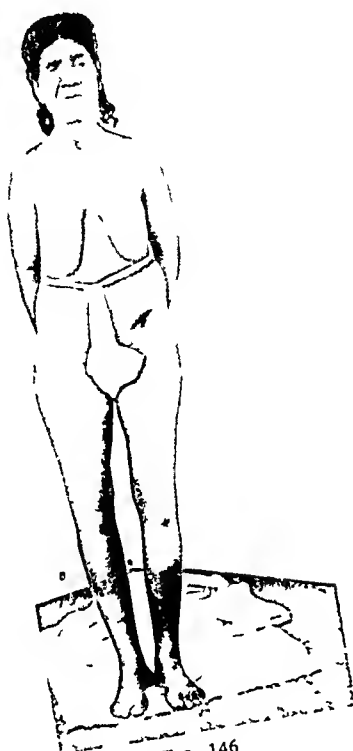


FIG 146



FIG 147

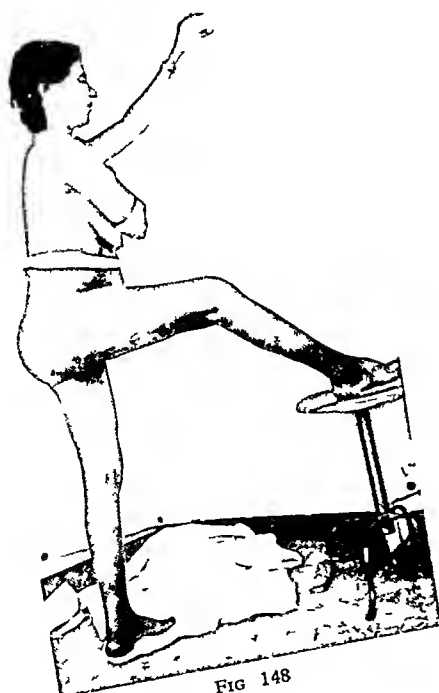


FIG 148

FIG 146—Case 1 The patient two years after operation

FIG 147—Case 1 Showing maximum abduction of the lower limbs

FIG 148—Case 1 Showing the maximum flexion of the hip possible in the injured limb two years after operation

FPA

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PART I

FRACTURES OF THE FEMUR WITH HIP-LUXATION
DUE TO ORDINARY TRAUMA

I FRACTURES OF THE HEAD OF THE FEMUR WITH HIP-LUXATION

Apart from "impaction within the limits of the head", of which Cotton has seen three cases, it is the rule for fracture of the femoral head to be associated with luxation of the hip. We have found thirteen cases of this association of injuries in the literature, and only one authentic case—recorded by Levin in this JOURNAL—in which an unimpacted fracture of the femoral head occurred without the luxation.

Sex and Age Incidence—Of the thirteen cases of head-fracture with luxation, eight were male and five female. The oldest patient was 60, and the youngest was 15. The age in one case is not stated. Only two patients were under 30. The average age was 38½.

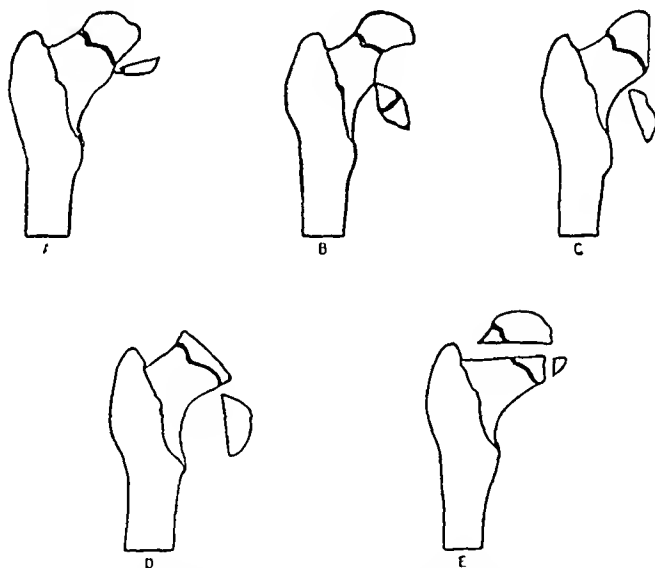


FIG. 151—Typical fractures of the femoral head. A, Braun's case, B, Crile's, C, Christopher's, D, Satta's, E, Riedel's (After Christopher).

The Hip-luxation—This is always in a backward direction. In seven cases it is described either as 'posterior' or 'dorsal'. In six the description makes it clear that the luxation was on to the dorsum ilii, in one case it was sciatic.*

The Fracture of the Femoral Head—Various fractures of the femoral head are illustrated in Fig. 151. In two cases, Riedel's and Nichiarelli's, the head was fractured in more than one place, and in one of these (Riedel's), the oblique fracture

* While the majority of authors agree in limiting the term 'dorsal' to luxations on the dorsum ilii, others employ 'dorsal' to signify 'backward' or 'posterior', and thus make the term 'dorsal' include the iliac, sciatic, and ischiatic luxations. It is sometimes impossible to discover from the records of femoral fracture with luxation whether the term is used in its wider or narrower connotation.

ran also into the neck. In one case (Robert's) the neck was separately fractured, but it seems probable that this fracture occurred during an attempt at reducing the luxation. In four cases a piece of the head remained in the cavity of the acetabulum, and in one it rested on its posterior lip.

Stimson's case is unique in presenting a groove on the anterior surface of the femoral head, parallel with and close to the edge of the articular cartilage, 1 in long, $\frac{1}{2}$ in wide, and $\frac{1}{8}$ in deep, along which the cancellous structure of the bone was exposed. This groove was made by an exostosis close behind the rim of the acetabulum. Stimson compares the injury to that occasionally produced in the humeral head by impact on the glenoid edge during the course of anterior luxation.

In four of the cases there was an associated fracture of the acetabulum—this, according to Tanton, is evidence of the manner in which the femoral head breaks (*see below* under 'Causation')

The Ligamentum Teres—In four cases this ligament is mentioned in two it remained with part of the head in the acetabulum. In Braun's case, where the fracture extended slightly into the neck of the femur (*Fig 151, A*), the ligament was split longitudinally. In the fourth case (Hinsdale's) it is merely stated that the fragment which lay on the lower margin of the acetabulum included the insertion of the ligamentum teres.

Causation—This is usually violence of a considerable degree. Three cases were due to train accidents, three to automobile accidents, one patient was thrown from a motor-cycle which fell on him. One fell from a cart, a sailor fell from the mast of his ship, a second fell through a hatchway, a woman fell 25 ft on to the pavement. In one case the nature of the injury is not stated, and in only one (Hinsdale's) does the trauma appear to have been slight.

The violence of the trauma was sufficient to cause the death of four cases, one dying of cerebral injury a few minutes after admission, another "soon after admission", and a third in three days with a fracture of the skull-base and of eight ribs, a fourth on the tenth day. Another case in which the luxation was reduced by manipulation probably belongs to the category of death from violence, since there was also fracture of ten ribs with hæmothorax. This patient died nine hours after admission.

The history of the single case due to slight violence is remarkable. This right-sided fracture with luxation occurred, writes Hinsdale, "in a woman aged 55, whose weight was about 160 lb. She fell in attempting to jump from a motor-boat to a wharf—she had reached forward to take a hand offered to her, and in jumping tripped over the gunwale of the boat, and fell on the wharf, striking her *left* hip. She was immediately conscious of a severe injury, and stated that she had dislocated the *right* hip-joint. She was unable to rise or move the limb, and was carried a short distance to her home" (We have italicized the words 'right' and 'left'). Hinsdale suggests that the contralateral luxation may have been due to the patient's catching her right foot on the side of the boat as she fell.

Christopher, in a study of this injury which has been our main source of information, adopts Tanton's view that the chief factor in these fractures is a force acting either in the long axis of the femur and towards its proximal end, or directly on the great trochanter—this force, they think, is suddenly applied in the course of the luxation in such a way that it breaks the femoral head against the acetabular rim, and this, according to Tanton, may also be fractured. "In all probability it

(the fracturing force) is always applied when the femur is flexed and slightly abducted" (Christopher). He discounts Birkett's theory that the pull of the ligamentum teres helps to break the head when the femur is driven upward and back, since, according to Cotton, the ligamentum teres is not strong and ruptures under a pull of 14 kilos.

Symptomatology —

Shortening — This is definitely recorded in four out of the thirteen cases, as 4 cm, 5 cm, 6 cm, and "about 3 in" (i.e., 7.5 cm), respectively.

Posture — Three authors report flexion, adduction, and internal rotation of the affected limb, one, flexion and adduction, one, flexion only, one, internal rotation only, one, adduction only, while in one case (Nichiarelli's) flexion was combined with *external* rotation—"The lower limb was rotated outward to such an extent that it lay with its lateral face on the bed." This rare condition of a dorsal luxation with eversion of the thigh was associated here with a fracture of the iliac lip of the acetabulum.

Movements — Nichiarelli obtained passive abduction to 45°, his patient could flex her thigh by sliding the foot along the bed. Other movements were absent. In Christopher's case slight extension was possible from the flexed position, in Satta's, passive movements were limited. No other authors deal specifically with movement.

Crepitus — This sign was elicited on rotation in only two cases.

Moxon found a large dent in the skin just above the left tuber ischi.

Diagnosis — According to Christopher, fracture of the femoral head with luxation is to be suspected in patients presenting the ordinary signs of a posterior dislocation of the femur when there is in addition crepitus in the neighbourhood of the joint or unusual difficulty in reduction. Cotton mentions ease of re-dislocation as another suggestive sign. A positive diagnosis cannot be made without either an X-ray examination, operation, or necropsy (Satta). In six cases the fracture was discovered by radiography, in one at operation, and in six others at necropsy, in none was the diagnosis made from clinical signs.

Treatment of Head-fractures with Luxation — In three of the thirteen cases under consideration no attempt at reducing the fracture or the dislocation is recorded. All three died soon after admission. (Robert's patient died, not from open reduction, but at once after an operation performed for resection of the femoral head, three months after the injury. He had almost succumbed during a previous unsuccessful attempt at 'closed' reduction under ether. Union of the fractured head had occurred in this case.)

Reduced Luxations — In nine of the thirteen cases the luxation was reduced, in six by non-operative means, and in three by open operation, two patients in the first category dying shortly after reduction, both were 'bad risks'. In Braun's case the luxation that was reduced by manipulation on the day of injury was complicated by hæmothorax and the fracture of ten ribs. Crile's patient died three days after admission with fracture of eight ribs and the skull-base.

Non-operative Reduction with Survival — Of the cases that survived, therefore, the luxation was reduced in four by 'closed' methods—in the case recorded by Platt, on the day of injury, in that of Christopher, after two days, in Nichiarelli's patient, after twenty days, and here partial union had occurred. In Durand's and Destot's case the interval between the injury and reduction is not recorded.

Platt's patient was first seen by him seven weeks after the luxation had been reduced by another surgeon. The fracture of the head was found by radiography, and a large piece of the head lying outside the acetabulum was removed from the joint through a posterior incision, after dividing the obturator externus. When seen by Platt the hip showed an almost complete range of mobility without discomfort. The primary object of his operation was to effect neurolysis of the sciatic nerve, which had been injured in the accident, the second was to remove the large separated portion of the head together with a slender fragment from the acetabular margin.

Nichiarelli mentions that he succeeded at the second attempt, "by the Paci-Lorenz method." Christopher is the only author who gives in detail the method of 'closed' reduction that he employed: "first, extreme flexion of the thigh, then extension to about 110 degrees, and then slow, steady abduction against considerable resistance. The fractured head slipped into the acetabulum with a crepitant sound. The motion in the joint at once became free." An X-ray after reduction showed the fragments of the head in excellent position.

Operative Reduction with Survival—These were three in number—Hinsdale's case operated on by E. G. Abbott on the first day after the injury, Satta's on the fifth day, Riedel's after three months. In Hinsdale's case and in Satta's, prior attempts at 'closed' reduction had failed. In Hinsdale's "the head could be placed opposite the acetabulum but would not enter. Crepitus was felt in these attempts." Radiography showed the fragment lying on the lower margin of the acetabulum. Hinsdale merely states that the luxation was reduced after removing the fragment and that the case was afterwards treated for three weeks by extension followed by movement. In Satta's case reduction had been twice unsuccessfully attempted before admission to hospital. After admission the luxation was reduced on a traction-table, but the reduction could not be maintained. Satta therefore, after removing the fragment consisting of half the head from the acetabulum, covered the raw extremity with fascia, nineteen days later the luxation recurred. Erysipelas and abscess followed a second arthrotomy.

Riedel merely states that he removed two fragments of the head and replaced what was left in the acetabulum.

Results—In the seven surviving cases of head-fractures with luxation, four good results are recorded—three after 'closed' reduction, and one (Hinsdale's) after operation (by Abbott). Thus Christopher's patient walked with crutches eighteen days after the luxation was reduced, and without them in thirty days. On discharge, fifty-three days after reduction, she could walk a mile a day, limping from slight foot-drop due to sciatic injury. Seven months later she could walk four miles without limping. Nichiarelli's patient, examined soon after removal of the plaster spica, showed negligible shortening. There was a moderate ability to flex the thigh, abduction was slightly limited by a varus deformity at the site of fracture, the gait was free with a very slight limp.

The hip-joint in Platt's patient preserved the good mobility which it had seven weeks after a 'closed' reduction, so that a year later hip-movements were complete and painless, though the gait was still imperfect owing to the sciatic lesion due to the original accident.

Hinsdale's case could walk two months after operation, and in four months was "perfectly well."

Ankylosis—This occurred in three of the surviving cases, who were aged respectively 15, 36, and 44. Riedel records in 1885 that when he removed the two fragments of the femoral head, three months after the injury, ankylosis of the hip occurred in the course of a further three months, accompanied, however, by only 2 cm of shortening. Riedel's patient was aged 15. In Satta's patient, aged 36, operative reduction was followed by erysipelas and an acute arthritis caused the ankylosis. Durand and Destot record a partial and painful ankylosis in a patient aged 44 who was treated by 'closed' reduction.

Comment—It is evident from the foregoing summary that good results can be got by non-operative reduction of the fractured and luxated head of the femur. 'Closed' methods therefore may be given a thorough trial.

Platt noted in his patient, who was aged 60, "the rapid restoration of excellent joint function, despite the presence of a large loose fragment", and it will be recalled that the fragment was afterwards removed incidentally, during an operation on the sciatic nerve, and not because it was causing disability. In Christopher's case, where a third of the femoral head remained in the acetabulum, "motion in the joint at once became free" after reduction. Nichiarelli, too, in his case aged 45, records "free mobility of the joint immediately after reduction".

Operation is thus indicated where 'closed' methods of reduction fail owing (1) to the patient's delay in coming for treatment (Riedel's case), (2) to an obstructing fragment (Hinsdale's case), or (3) as in Satta's case, where the femoral head was fractured at a right angle to the long axis of the neck (*see Fig 151,D*), on account of the impossibility of maintaining reduction either of the fracture or the luxation.

The longest interval between the injury and the successful reduction of a dislocation, which gave a good result, was twenty days (Nichiarelli's case).

Good results by 'closed' methods have been obtained whether the fragments of the head are displaced outside the acetabulum (Platt and Nichiarelli) or remain within (Christopher).

II FRACTURE OF THE NECK OF THE FEMUR WITH HIP-LUXATION

Besides our own case (*Case 2*), we have found records of fifteen others in which it is claimed that this association of injuries was caused by ordinary trauma. Two of these sixteen cases, Robert's and Riedel's, have already been dealt with, in Robert's case there was also a separate fracture of the femoral neck, and in Riedel's the oblique fracture of the head ran through to the upper part of the neck (*see Fig 151,E*). In three of the sixteen cases—Chitwood's, de Chegoïn's, and Harcourt's—the records do not clearly establish a diagnosis of femoral fracture with luxation; all three are rejected by Stimson*. Eleven cases out of the sixteen are thus left for study.

* These three cases have been quoted as authentic by various writers, we therefore summarize them here—

H de Chegoïn's case, seen in 1868, is reported by Wippermann, who gives the following details. A female, aged 77, was injured by falling from her chair. The case was diagnosed as a fracture of the femoral neck with an unspecified luxation of the hip. The injury was treated by slight extension only. Sixteen months later the great trochanter was found to be displaced upwards and back; there was shortening and eversion of the foot. Five years after the injury she could go up and down stairs. Stimson believes that this was a case of simple fracture of the femoral neck.

In Chitwood's case, a male, aged 45, a diagnosis was made of luxation on to the dorsum

Sex and Age Incidence.—Of these eleven cases seven were male, one was female in three the sex was not stated. The oldest was either our own case, who did not know his age but looked 70, or Monk's, aged 64, the youngest was the female patient, aged 13. The average age of the eleven patients was 37, five of them were over 50.

The Hip-Luxation with Neck-fracture—In six cases the luxation was backward, in two pubic, while in one (our own *Case 2*) it was downward and forward, in two cases the variety is not recorded.

Causation—Great violence in which it is impossible to distinguish separate or successive elements caused four of the eleven fractures of the femoral neck accompanied by hip-luxation—the fall of a building, a fall from a roof, the impact of a tramcar, an automobile, overturned by a train, pinning down the victim. Our own case, on the other hand, merely slipped on the footpath (he thought on banana peel) with his lower limbs apart.

Tunnecliff and Lossen each record the clear succession of two separate traumatic events †. In Tunnecliff's case, where the injury was on the right side, a tree fell on the patient's left shoulder driving his feet through a sheet of ice. His body was inclined to the right, and, as he fell through, the right great trochanter struck hard on the edge of the hole. In Lossen's case a ladder crashed with the patient, who landed on his feet, and then fell sideways, striking the great trochanter of the affected limb against the ladder.

Post's case, a girl of 13, received a blow on the back "with a twisting of the body to the right and the lower extremities to the left." Both hips were luxated and the left femoral neck was fractured. The definite history given in the two cases just quoted, suggests the possibility of an unrecorded fall on the left side in Post's patient.

In the remaining three cases of this series of eleven, the cause of injury is not stated.

Neighbouring Fractures—In only one case (Buchanan's) was there evidence of a neighbouring fracture. "the outline of the pelvis near the acetabulum was indistinct as though from a fracture" in an X-ray taken five months after the accident.

Local Complications—In Post's case there was necrosis of the luxated head of the femur. This complication of hip-luxations was first noted by Sir Astley Cooper. "Sometimes, after the reduction of dislocations, suppuration ensues, and the patient falls a victim to excessive discharge and irritation." He quotes two examples with death occurring in a few days. (Two instances of sepsis following 'closed' attempts at reduction will be found in *Part II*.)

III, with a fracture of the femoral neck, which Kammerer believed was only partial. The injury was caused by a fall from a wagon which then unloaded its contents on the patient. The fracture was not diagnosed by Chitwood but by the physician in charge, five days after the luxation had been reduced by manipulation. The fracture was then complete—Kammerer thinks, as a result of the reduction.

In Harcourt's patient, a male, aged 41, the presence of a luxation "probably forward on the pubis" was diagnosed according to Kammerer solely from a snapping noise heard while the case was being examined under anaesthesia.

Stimson characterizes the diagnosis in the last two cases as "wholly untrustworthy."

† Kammerer believes that every case of hip-luxation with femoral fracture is "the result of two distinct violences", and he cites the cases of Tunnecliff and Lossen, adding however Chitwood's, to which we have already referred.

Buchanan's patient suffered from peroneal palsy which is referred to below under the heading 'Treatment'

Treatment of Neck-fractures with Hip-luxation.—Three of the eleven cases (Haase's, Gartner's, and Douglas's) received no treatment, the fracture and luxation being found post mortem. In our own case the proximal fragment of the fracture was so completely separated and so far displaced that we thought its reduction imprudent in a frail and aged patient, and we removed it at the operation because its sharp end was thrusting the profunda vessels forward. Post's case was not reduced, and Lossen makes no mention of reduction. The cases where reduction of the dislocation was effected number only four—three, of which one died, were reduced by 'closed' methods and one by open operation.

'Closed' Reduction.—Monks reduced an anterior luxation somewhat like that present in our own Case 2, except that the head lay higher and was turned proximally instead of distally—the fractured end of the proximal fragment projected forward, causing venous obstruction and stretching the superficial femoral artery over it. Fearing that this fragment would ultimately open the vein or artery, Monks decided to attempt immediate reduction, and in case of failure to excise the luxated head. Under ether anaesthesia "steady traction was made by an assistant while an attempt was made to manipulate the bone back into the joint. This was successful, but only after a prolonged trial during which the leg was placed in a variety of positions." The patient died seven hours after admission; he had, in addition to his femoral fracture and luxation, a fracture of the clavicle and of four ribs in the cardiac area.

Tunnecliff records (1868) one of the two cases of 'closed' reduction that survived in this group. The patient was left for thirty-eight days until there should be a measure of bony union in the fracture*. The sciatic lesion was then reduced by flexion and abduction of the thigh, with direct pressure on the femoral head. As the head crossed the acetabular rim the neck of the bone was re-fractured. A long external splint was applied to the limb and extension made through a foot-piece, with counter-extension from the perineum. Five weeks later the patient could walk with one crutch, and afterwards progressed favourably with only $\frac{1}{2}$ in of shortening.

Hoeftmann records (1904) the second case of survival in this group after reduction by a 'closed' method. His patient was a male with posterior luxation of the hip and a fracture of the femoral neck, both on the right side—the patient's age is not stated. Many attempts at reduction failed, and ten weeks after the accident, a fall from a roof, X-ray examination showed that the head of the femur had united almost exactly at the *back* of the femoral neck. The limb was markedly rotated inwards and was adducted. When the posterior fixation of the head was realized "the reduction was child's play", the limb was rotated outwards till the foot pointed backwards, when the femoral head at once slipped into the acetabulum.

* Tunnecliff, who saw his case a month after it had been diagnosed by Drs Woodworth and Lord, writes "I must confess that I had at first some doubt about the alleged fracture, but Drs W and L both assured me most earnestly that they could not be mistaken on this point, as both had felt distinct crepitations on their first visit, and then the great mobility of the limb at that time would seem to confirm the view. I observed further that pressure upon the neck of the bone at the base of the great trochanter elicited evidence of tenderness at that point, and I was forced to acquiesce in the views of the attending physicians."

The foot, however, still pointed back after the reduction was effected. The femur therefore was sawn through just below the trochanters in order to correct this deformity.

Operative Reduction—The only operative success in this group belongs to Buchanan, of Pittsburgh. He first saw his patient, when the fracture was already united, five months after the accident. Prolonged extension was disappointing, and the case was treated by open operation some seven months after the original injury. The acetabulum was exposed through Kocher's incision; it was filled with dense connective tissue, which was dissected out, exposing the normal cartilage. Traction was applied through adhesive plaster strips attached to the flexed thigh, by means of a strap passing round the shoulder of an assistant and hooked at its ends into rings attached to the plaster strips. The lifting effect of the shoulder-traction was supplemented by the assistant hooking his elbow under the patient's knee, standing, as he did so, on a stool. Four levers were then passed under the femoral neck, and by combining manipulation with traction the head was reduced. The patient recovered from the shock of the operation and from reactionary oozing that occurred later in the day. The limb was splinted in abduction, and extension was applied to the other limb "to keep the pelvis from tilting towards the injured side." After operation, foot-drop was noticed, which persisted for several months, but finally disappeared; it was thought to be due either to pressure on the sciatic nerve by the levers, or more probably to the traction exerted on the external popliteal at the bend of the knee.

A case reported by C. E. Thomson, though it does not belong to *Part I*, since the fracture was caused by surgical intervention, bears on the operative treatment of this condition. The femoral neck was broken in an unsuccessful attempt to reduce an unspecified luxation of the hip of eight months' duration in a male whose age is not stated. Ten days after this surgical accident the luxation was reduced by open operation: anterior and posterior incisions were made, and the femoral head was replaced by traction with blunt hooks. Reduction of the fractured neck was maintained by means of a specially devised nail, and the author concludes his account with the hope that the patient will have a "useful stump". (The leg on the side of the fracture and luxation had been amputated five weeks after the original accident.)

Results—Only three good results have been definitely recorded in neck-fractures with luxation—Tunnecliff's (1868), and Hoeftmann's (1904), treated by 'closed' methods thirty-eight days and seventy days respectively after the injury, and Buchanan's (1920) by open operation seven months after the injury.

Results in Cases Successfully Reduced—Tunnecliff's case could walk with one crutch five weeks after reduction; the subsequent progress was favourable, and the shortening amounted only to $\frac{1}{2}$ in. In Hoeftmann's case the result is recorded as 'satisfactory'. Buchanan's patient was fitted with a brace seven weeks after operation, and a week later walked with it in the ward, still showing signs of a partial peroneal paralysis. Five and a half months after the operation, when he discarded the brace, there was no shortening but some eversion, which was attributed to the destruction of the internal limb of the Y-shaped ligament. Six and a half months after operation the peroneal palsy had disappeared and he walked with comfort; shortening of from $\frac{1}{2}$ to $\frac{3}{4}$ in was now evident, and eleven months after operation this had increased to $1\frac{1}{2}$ in. He then walked

"with a solid gait and without pain and has for some months been working as an automobile repair man" *

Monks' case with multiple injuries who died a few hours after a 'closed' reduction has been referred to on p 214

Results in Umeduced Cases—In Post's case the proximal fragment was excised for necrosis causing a sinus six months after the accident, there was a final shortening of $4\frac{1}{2}$ in (The other hip, which had also been dislocated in this patient, was successfully reduced with full restoration of function in that hip) The result is not mentioned in Lossen's unreduced case, while the cases of Douglas, Haase, and Gartner were investigated post mortem

Our own Case 2, after excision of the proximal fragment and insertion of the remainder of the neck into the acetabulum, insisted on leaving hospital while in full abduction in a plaster-casing which he broke to pieces in a few days. He was seen later, wearing fragments of the casing, with 4 in of shortening, but he refused all further treatment

Comment—It will be noted first that in the three cases where good results were obtained (Tunnecliff's, Hoeftman's, and Buchanan's) union of the fractured femoral neck had occurred before the hip-luxation was reduced. Kammerer, in some experiments on the cadaver which are relevant to this point, found that, if the *shaft* of the femur were broken, he was able to reduce a coexisting luxation of the hip by grasping the end of the proximal fragment with lion-forceps through an incision, and manipulating the head through the skin. Fracture of the *neck*, on the other hand, prevented him from reducing the luxation. We have since found that Richet anticipated Kammerer's experiments in 1853. Richet, unlike Kammerer, easily reduced luxations of the hip associated with neck-fracture in the cadaver simply by pushing the head back into place.

These results, in apparent contradiction, led us to repeat the experiments. Our attempts were confined to posterior luxations with fracture. If the proximal fragment was large, as in shaft-fracture or *distal* fracture of the neck, we could easily reduce the luxation by direct pressure applied to the femoral head, with the subject prone in the Stimson position—the lower limbs hanging over the table at right angles to the trunk. If, however, the fracture was subcapital and the proximal fragment was small, we could not reduce it. By passing a finger through a stab-wound we found that failure was due to the difficulty of guiding a small fragment, which slipped and turned turtle, back through the collapsing tunnel torn in the soft tissues by the luxation. We think that our experiments perhaps explain the discrepancy between those of Richet and Kammerer.

In Thomson's case, quoted above, of surgical fracture with a hip-luxation eight months old, operative reduction was accomplished and the result promised well, though the fracture had not previously united.

One problem for the surgeon, then, is whether (1) to wait for union of the fractured neck before attempting to reduce the luxation, or, (2) to operate early, before union has occurred, while the luxation is still recent. Decision is less difficult in cases like our own, where the proximal luxated fragment is so widely divorced from the rest of the femoral neck that it would be impossible to reduce

* We were not able to ascertain whether De Morgan reduced the luxation in his case or not, if he did, he used a 'closed' method. The patient walked with a stick twelve weeks after the injury and returned to work.

it, and where (as in Monks' case and in ours) the proximal fragment is a danger to important vessels. Dangerous fragments must be removed, and the opportunity will then be taken of thrusting the distal part of the neck into the acetabulum—elongating the neck in case of need by Whitman's method of detaching the great trochanter and re-affixing it lower down the shaft.

It is unlikely, in older patients, whose uncomplicated fractures of the femoral neck so often fail to unite, that the chance of union will be increased by cutting down to reduce the luxation, *unless the fragments can be artificially impacted*.

In younger patients suffering from grave additional injuries, who by the time their dislocation is 'old' have recovered sufficiently to undergo operative reduction, the interval of waiting must be employed in securing proper union of the fracture. The fragments should be adjusted under radiographic control, for, as Kammerer first pointed out, mal-union of the neck may be such that when the luxation is reduced the patient is crippled by eversion of the limb—a fact strikingly exemplified in Hoeftmann's case, where ten weeks after the accident the proximal fragment bearing the femoral head had become united to the *back* of the neck. It will be recalled that after the luxation had been reduced Hoeftmann was obliged to divide the femoral shaft to correct backward pointing of the foot.

Suggested Plan of Treatment in Neck-fractures with Hip-luxation —

The treatment of fractures of the femoral neck is often difficult when they are uncomplicated, and it would be premature—on a basis of only three good results—to do more than sketch a provisional plan for treating them when complicated by luxation. Certain eventualities are here put forward, and the treatment of each is suggested.

1 *The Proximal Fragment Endangers the Main Vessels*—Operate at once to free the vessels, dealing with the proximal fragment according to the circumstances specified in the following paragraphs.

2 *The Proximal Fragment is merely Divorced from the Rest of the Femoral Neck*—

a In *the aged*, but apparently robust, where useful union is unlikely, the fragment may be left alone and an attempt made to thrust the rest of the neck into the acetabulum, and to maintain it there by fixing the limb in abduction and inversion. The neck may be lengthened in case of need by detaching the trochanter major and re-affixing it lower down the shaft.

b In *younger patients*, open operation will give an opportunity of replacing a completely separated head. We think that Cotton's method of artificially impacting the two fragments by mallet blows on the great trochanter may then sometimes solve the problem of union. (One of us (M. B.) has used artificial impaction of the femoral neck with success in an open wound.) We feel, too, with Cotton, that to peg-graft a head which has been so completely separate is to place undue reliance on the union of two devascularized pieces of bone.

3 *In Patients Suffering from Other Grave Injuries—of Abdomen, Chest, or Skull ('Poor Risks')*—

a *Those who recover as 'good risks'*. Fix the fracture in good position and reduce the luxation either by a 'closed' method or by open operation after bony union has occurred, remembering that Tunnecliff reduced a luxation successfully on the thirty-eighth day by manipulation, and Buchanan by open operation after seven months.

b Those who continue as 'poor risks' Fix the fracture in good position and compensate the inevitable shortening by a suitable prosthesis

4 *Neglected Cases where Bony Union of the Fracture has Occurred*—

a In good position (as regards the relation of the head and neck)—These should be treated, when possible, by open reduction (Buchanan's case is an encouraging example)

b In bad position, so that after reduction there will be, perhaps, a disabling or disfiguring eversion of the limb These cases may require osteotomy of the femur—usually of the shaft (as in Hoeftmann's case)—and re-adjustment, after the luxation has been reduced Softening or deformity of a head long luxated may, however, call for resection and implantation of the neck into the acetabulum *

5 *Neglected Cases without Union of the Fractured Neck*—Must be operated on in 'good risks' whenever possible, reducing the luxation and impacting the fracture, or, where this cannot be done, implanting the neck into the acetabulum In 'poor risks' the treatment will be as in paragraph 3 (*b*) above

III FRACTURE OF THE SHAFT OF THE FEMUR WITH HIP-LUXATION

In addition to our own *Case 1*, we have collected fifteen records of this injury from the literature †

Sex and Age Incidence—Only two of these sixteen patients were female The age incidence is somewhat remarkable—8, 9, 9, 12, 13, 13, 14, 16, 19, 24, 38, 40, 51, 64, 'adult' (the age in one case is not stated) Thus more than half the cases are under 20, the average age being 21 Our own case, aged 64, is the oldest of the series

The Luxation of the Hip-joint—This was reported either as 'posterior', 'dorsal', or 'upon the dorsum ili' in eight cases (see footnote on these terms, p 208) In two cases it was stated to be sciatic, in one, downward and backward between the acetabulum and the ischial tuberosity Pubic luxation occurred in two cases, suprapubic in one, and obturator in two There does not appear to be any significant association between a particular type of luxation and the level of the fracture in the shaft

The Fracture of the Femoral Shaft—The level of this fracture is recorded in fourteen cases, in all but two it lay either in or above the middle third of the bone thus one fracture was in the lower third and one at the junction of the lower and middle third, while four were in the middle third, four at the junction of the middle and upper thirds, two were in the upper third itself, and two were subtrochanteric In Delagarde's case the shaft was fractured in two unspecified places

* An unintentional fracture of the femoral neck has re-united (Tunnecliff's case), but re-fracture of the neck is not to be recommended unless the head-fragment has become fixed in a position that makes it impossible to reduce the hip-luxation

† Unless we include the case that Markoe reported in 1855 This patient, a boy, aged 8, fell from a high ladder and was believed to have a posterior luxation of the right hip, with compound fracture of the right femoral shaft The case was seen and treated by Markoe's resident medical officer The right foot was stated to lie in the left axilla the limb was brought back across the abdomen and was adducted and lowered to the bed Reduction of the luxation is said to have occurred during this manœuvre There was also a simple fracture of the left thigh Hamilton was not convinced by this account The case was reported as cured

Causation—We have already spoken of Kammerer's belief that the association of any femoral fracture with hip-luxation is always the result of at least two separate traumatic events, but in most of the cases in this group it is easier to suspect than to distinguish them. In seven of these sixteen cases with shaft-fracture, the injuries were due to gross violence in which it is impossible to find either sequence or distribution—the fall of coal, of a wall, of chalk in a chalk-pit, of a tree, a crush by a cart-wheel, impact with a tramcar. Allis's case, though struck by a moving railway-car, was hit only on the shoulder, no other detail is given of the accident. Bainbrigge's patient, a woman aged 38, and an invalid for two years, was standing with her left hand on a table, her right on a cane. Losing her balance, the weight of her body was thrown on the left leg, on which she made a semi-revolution and then fell to the ground. O'Connor's patient fell from a height of ten feet to a beam which passed between his legs, he then fell fourteen more feet to the ground. Kammerer gives a very full history of the case in his boy-patient, aged 13, but we are unable to share his confidence in assessing separate causes for the fracture and luxation, since he states that the boy felt no pain when he was first thrown backwards from a swing that carried six children, nor yet while he was trying to escape from its path; the swing, however, on its return, struck the thigh so heavily at the site of fracture that the boy fainted. The right foot of Cook's patient slipped into a hole, and he fell with his legs wide apart. When trying to rise with his foot still caught, he fell again, on his right side. Murdoch's patient fell between railway trucks, and was struck by the brake-rods as two cars passed over him. In Fergusson's case and in Birkett's the cause is not stated.

The cause in G. R. Harris's case was peculiar. D. H., a boy aged 9, was running over heavily crusted snow, when he broke through and fell forward.

Another boy, who was running after him, fell on top of him. The patient was unable to arise and was carried home. (In this case the luxation was into the obturator foramen, and the shaft broke at the junction of the middle and upper thirds.)

Thus in seven cases there is a record of great and indiscriminate violence. In six there is a suggestion of two events, a wrench and a fall in Allis's*



FIG 152—Illustration of the way in which crushing loads may produce hip-luxation. The flexion, adduction, and rotation inwards may here luxate the right hip. A fall or impact may then break the right femur. This combined mechanism is relevant to certain cases described in the text. (Re drawn from Cotton.)

* It is possible that the causation in our own case resembles that in Allis's patient—a wrench due to the tram's impact and a fall on the street, but we could get no clear history from the patient.

(Fig 152) and Bainbrigge's cases, forced abduction and secondary falls in those of Cooke and O'Connor, and a primary fall with a secondary impulse in Harris's case, and possibly, too, in Murdoch's

Treatment and Results of Shaft-fracture with Hip-luxation—In eight of the sixteen cases the luxation was reduced. No attempt at reducing the luxation was made in four of the seven cases in which it was not reduced, though in each the fracture was treated. The dorsal luxation was not diagnosed in Allis's patient for fourteen weeks (1879), and in Bainbrigge's (1846) for five months, after the accident. Murdoch (1878) found a posterior luxation in his case post mortem. Kammerer (1889) comments as follows on the pubic luxation which he first saw ten months after the injury: "A reduction of the dislocation would, I fancy, cause an inward rotation of the lower limb of about 45° , and would certainly greatly impair its usefulness, unless the bones were refractured and set in a correct manner. And I think that no valid objection could be raised to such measures. In my own case, then, it is only the impossibility of reduction which has prevented the attempt. When I saw the patient the dislocation was of ten months' standing."

It should be observed that the most recent of these abstentions from reduction was recorded as far back as the year 1889. In one case, Thornhill's, the fact of reduction is doubtful.

Failures—In three cases reduction was attempted and failed. Delagarde's case of sciatic luxation with double fracture of the femoral shaft was of five months' standing when first seen. The fracture had united, and he excised the unreduced head. The patient is described as 'relieved'. In Fergusson's patient (posterior luxation and fracture of lower third of shaft) the luxation was not detected "for some considerable time", the shaft had united. In Forster's case (dorsal luxation and fracture of middle third of shaft) the luxation was not reduced.

Successful Reductions of the Luxation with Shaft-fracture—These were obtained in eight of the cases in this group: seven were effected by 'closed' methods, one by open operation, no case died.

Sir Astley Cooper, in 1823, recorded the first successful treatment of the condition "hip-luxation with ipsilateral fracture of the femur" in a case belonging to this group with shaft-fracture. "The hip was dislocated on the dorsum and the same thigh broken about the middle of the bone. As the reduction of the hip was, of course, impracticable, the thigh was bound up in the usual manner and treated without any reference to the dislocation of the joint, with the hope that, when the thigh-bone was reunited, the hip might possibly be reduced. At the end of five weeks, the bone appearing tolerably firm, I had a very careful and unremitting extension made by pulleys, and in less than half an hour had the satisfaction of feeling the head of the bone re-enter the socket. The patient became so upright as to show scarcely any signs of lameness afterwards."

The pubic luxation in Bloxham's patient, aged 14, was also reduced by pulley-traction, but before the shaft had united. The fracture here was just above the middle third. When a week's immobilization in short splints had reduced the swelling, the entire limb was fixed in long splints. The pelvis was fastened to the table, traction was then applied from a pulley fixed to the ceiling one foot to the right of a point vertically above the patient's navel. Reduction here too occurred in thirty minutes under the resultant of (1) the vertical pull, (2) the pull of the gluteal muscles, and (3) direct manipulation of the femoral head. Passive

movements were begun eight days after reduction, and eight months later the patient had $\frac{1}{2}$ in. of shortening with full function

Two cases, Etève's (posterior luxation and fracture of middle third of shaft), and Cooke's (obturator luxation and subtrochanteric fracture), aged 8 and 9 respectively, were reduced, one day and "at once" after injury, by direct pressure on the femoral head, supplemented in Etève's case by hip-flexion to 90° . The result in Etève's case is not stated by Malgaigne, who quotes it, but Cooke's patient, treated on a double-inclined plane for fifteen days after reduction, and then with a long external splint, had no shortening and limped only a very little. There was, however, a good deal of thickening at the site of fracture.

O'Connor's case (dorsal luxation and fracture of junction of upper and middle third of shaft) is remarkable in that the traction which reduced the fracture is said to have reduced the luxation at the same time, owing, it was thought, to spasm of the vasti. Neither the patient's age nor the interval between injury and reduction is stated. The man recovered with a $\frac{1}{2}$ in. of shortening.

Bryant states that in 1860 he saw Birkett reduce a pubic luxation associated with subtrochanteric fracture in a boy aged 12 "by gentle well-directed movements", no further details are given.

G. R. Harris, who most kindly sent us details of his case, reduced an obturator luxation associated with shaft-fracture at the junction of the middle and upper thirds in a boy aged 9. Making the father fix the pelvis, Harris grasped the femur above and below the fracture, and reduced the luxation by rotation and extension. The after-treatment was by extension with strapping and a weight of 10 lb., while the thigh was fixed between sand-bags. The boy was up and on crutches in four weeks, three weeks later there was a $\frac{1}{2}$ in. of shortening, and in six weeks after the accident he was climbing trees.

Our own Case 1 (female, aged 64, luxation on dorsum ilii and fracture of upper third of shaft) is the only one in this series whose luxation was reduced by open operation. The method of reduction, three weeks after the accident, has been sufficiently described in the introduction to this study.

Comment.—Except in Murdoch's patient, who died a year after the injury, from an operation on the *fracture*, and in whom the luxation was found post mortem, there were no deaths in this group of sixteen cases. The age of six of the seven cases with luxations reduced by 'closed' methods was between 9 and 16 years, the seventh case (O'Connor's) is described as 'adult'. The interval between the accident and reduction in four of these seven cases was short, varying from 'immediate reduction' to reduction after eight days. Only in one case (Sir Astley Cooper's) had the fracture united prior to successful reduction of the luxation, union occurring in a five-week interval after the accident. The intervals in Birkett's and in O'Connor's cases are not mentioned.

Ease of reduction is definitely related to weakness of musculature, a fact noted by Richard Wiseman, Serjeant-Chirurgion to Charles II, in 1676. This probably accounts in some measure for the successful cases in this group where so many of the patients are children.

The cases in which reduction either was not tried or failed when attempted were all 'old' luxations, fourteen weeks being the shortest interval recorded between the accident and the recognition of an 'old' luxation or the attempt to reduce it.

In our own case resort was made to operation three weeks after the accident, when two attempts at 'closed' reduction had already failed

Suggestions regarding Treatment of Shaft-fractures with Hip-luxations—The successful cases quoted above encourage a trial of 'closed' methods at the earliest possible moment, splinting the fracture, and treating the luxation by direct pressure on the femoral head. Our own experiments on the cadaver lead us to think that in the case of *posterior* luxations this pressure can be applied with most effect and with least fatigue when the patient is in the Stimson position, lying prone with his limbs hanging vertically over the end of the table. When the luxation is *pubic*, pressure can be applied when the patient lies on his back. The muscular relaxation obtained by spinal anaesthesia should greatly facilitate reduction under these conditions.

Even in cases of some standing the surgeon may take heart from the fact that Sir Astley Cooper, in a boy of 16, reduced "in less than half an hour" a luxation on to the dorsum ilii with pulleys after five weeks, when the fracture at the middle third had united.

Operative Reduction of Shaft-fractures with Hip-luxation—Our own case showed us that excellent exposure both of the luxation and the fracture can be got by the well-known Smith-Petersen approach combined with the anterior exposure of the femoral shaft to which we have already referred. The proximal fragment might be manipulated with a special fracture-hook, or, as in our case, with a strong wire transfixing the bone and twisted into a wide ring for comfortable traction. With this incision the femoral head can easily be controlled and levered into the acetabulum.

Hey Groves has pointed out that in difficult cases of old-standing congenital luxations of the hip, open reduction may become easy after dividing the femoral shaft near its upper third, a step which eliminates the pull of many muscles that oppose the descent of a proximally displaced head. Hey Groves thus confirms on the living the experiment performed in the dead by Kammerer some fifty years back.

Cases of Shaft-fracture with Luxations Long Unreduced—The femoral head in these patients may be so deformed by pressure that it cannot be usefully replaced, it must then be resected in those fit for operation, implanting the neck into the acetabulum after lengthening it in case of need by Whitman's method.

Cases that are Irreducible—Finally there are those cases in which from any cause reduction of the luxation is impossible, or is contra-indicated. The fracture none the less should receive most thorough treatment. As a rule there is a progression of the immediate shortening, and in only one case (Kammerer's) is a patient with his luxation unreduced reported as having satisfactory function. This boy, though unable to move his hip-joint "could walk and run very little hindered in the activities of life." The majority of these patients, however, despite the formation of new sockets for the head, will be rendered unfit, like Fergusson's case, for "active pedestrianism."

IV FRACTURES OF THE FEMUR OTHER THAN OF THE SHAFT, NECK, OR HEAD, WITH HIP-LUXATION

We have found only three undoubted cases belonging to this category. Borchard saw a boy, aged 14, in whom suprapubic luxation of the hip was accompanied by a *fracture of the great trochanter*, and mentions a similar case seen by

Lauenstein The injury in Borchard's case was due to a fall under a roller drawn by a runaway horse The tibia and fibula were also fractured, and the patient died soon after admission to hospital *

Drehmann, in 1896, reported a case of von Mikulicz, with a left dorsal hip-luxation and an *epiphysial separation* of the left femoral head the boy, aged 8, fell to the ground while carrying another boy on his back Immediate efforts at reduction failed After fourteen days in bed the boy came limping to hospital on the toes of his left foot with the knee and hip bent All hip movements were limited The femoral head was felt posteriorly, close to the great trochanter, when the thigh was moved There was 4 cm of shortening A further unsuccessful attempt at 'closed' reduction was made in hospital, ten weeks after injury Von Mikulicz then separated the great trochanter through a transverse incision and evacuated the acetabulum The epiphysial separation which makes the case unique was found during operation The epiphysis was displaced posteriorly and outwards Though union is not mentioned, the fragment had probably united with the neck in the ten-week interval before the operation, since (1) the position of the head was first recognized by moving the thigh, and (2) some degree of external rotation persisted in spite of attempts at correction Von Mikulicz reduced the dorsal luxation by manipulation, and sutured the trochanter back into place, splinting the limb in extension and outward rotation The wound healed by first intention, and the patient walked with a stick in six weeks, leaving hospital three months after operation This case is probably comparable with that of neck-fracture with luxation, recorded by Hoeftmann (*see above*), where the posterior fixation of the proximal fragment caused extreme eversion of the limb after reduction, which was only corrected by dividing the femoral shaft

SUMMARY OF THE GROUPS OF FEMORAL FRACTURE WITH HIP-LUXATION DUE TO ORDINARY TRAUMA

We can now compare certain features of the various categories The number of cases in each group is too small to establish etiological factors of decisive value, but certain points are suggestive

Age.—Fractures of the *shaft* with luxation, claim the six youngest patients, (8, 9, 9, 12, 13, and 14 years), and this group is the only one in which more than half the recorded cases are under 20, the average age in the group being 21 Among the femoral *neck* and *head* fractures, only three cases, and one case, respectively, are under 20 years, the average age in each of these two groups being 37 and 38½.

Sex.—Only eight females have been found with femoral fracture and hip-luxation (eight out of forty-two authentic cases) † Five of them occurred in the

* A case of hip-luxation with fracture described by Monks in a man aged 60 is omitted as Monks is doubtful whether the fracture was of the great trochanter or of the acetabulum

† Gaub's case (1917) which was communicated privately to Buchanan, who quotes it (*loc cit*, Case 49, p 147), is not included it has been impossible to classify as we were unable to learn the site of the femoral fracture The patient, a male aged 35, was rolled between railway cars, sustaining an 'anterior' luxation of the hip, which was overlooked for three months in the presence of other injuries After clearing the acetabulum Gaub reduced the luxation through an anterior incision by means of levers and traction, and applied a plaster case extending to the nipples The functional result was excellent, the patient now working as a conductor It is not stated whether the femoral fracture had united before the reduction

group of thirteen patients with fracture of the femoral head. Only one female suffered from fracture of the femoral neck, and two from shaft-fracture.

Relations of the Variety of Fracture to the Variety of Luxation—The following table, modified from Kammerer's, shows this relationship—

		FRactURE OF HEAD	FRactURE OF NECK	FRactURE OF SHAFT	OTHER FEMORAL FRACTURES	TOTALS
<i>Luxation backward</i> (30 cases = 75 per cent)	Described as 'dorsal', 'posterior', or 'upon dorsum illi'	12	4	8	1	25
	Into sciatic notch	1	1	2	—	4
	On to ischial spine	—	1	—	—	1
	Behind lip of acetabulum	—	—	1	—	1
<i>Luxation downward</i> (4 cases = 10 per cent)	Into obturator foramen	—	—	2	—	2
	Down and forward into Scarpa's triangle	—	1	—	—	1
<i>Luxation forward and up</i> (6 cases = 15 per cent)	Pubic	—	2	2	—	4
	Suprapubic	—	—	1	1	2
		13	9	16	2	40*

* In two cases, both with neck-fracture, of the forty-two cases reckoned as authentic, the variety of hip luxation is not specified.

Injury Causing the Luxation and Fracture—Fractures of the femoral head appear to have been associated with the greatest violence—sufficient in four cases to cause early death—only one case in this group was due to relatively slight trauma. The possibility of the luxation and the fracture being "the result of two distinct violences", as Kammerer believed, has been fully discussed under the headings of neck- and shaft-fractures.

Symptomatology—This has been dealt with above only in the group with head-fractures, where it has been treated as fully as the records permit. In the other groups information on this point is scanty. It is best summarized in the words which Stimson adapts from Koch: "When the two injuries (femoral fracture and hip-luxation) have coexisted the diagnosis has sometimes been made by recognizing that the head, which could be felt out of its place, did not share in the movements communicated to the shaft."

Results of Treatment—In the series of forty-two authentic cases the hip-luxation was reduced in twenty-two patients, in sixteen by 'closed' methods with eleven good results, in six by operation with four good results. (Thornhill's doubtful 'closed' reduction is not included, and in one case of 'closed' reduction the result is not stated.) In fifteen cases, therefore, the functional result was good* after reduction, while in six cases where reduction was effected the result was disappointing. Thus three patients died after 'closed' reductions. One case

* 'Good', that is, as regards the hip, three of these cases were complicated by sciatic lesions, one of which admittedly followed operative reduction, and this was the only one which had recovered when the cases were reported.

of ankylosis occurred after operation, and one after 'closed' reduction. In one case the luxation recurred after a first operation and sepsis followed the second arthrotomy.

The worst results were obtained when the femoral *neck* was fractured, four luxations were reduced out of ten cases. Of these, three were reduced by 'closed' methods (one patient dying soon after of multiple injuries), one was reduced by operation. In the three surviving cases the fracture had first been allowed to unite, so that the femoral lever was re-established. Drehmann, in the equivalent condition of epiphysial separation of the femoral head, obtained a good result by operative reduction ten weeks after the accident, union of the fracture had probably occurred.

The want of success in this group is repeated in *Part II*, where the femoral neck-fracture was caused by attempts to reduce a luxated hip, and here we find a satisfactory final result in only one case out of seventeen.

More luxations were reduced in association with fracture of the femoral head than with any other fracture. This is natural since the femoral lever in this fracture remains unbroken, unless the fracture is transverse to the long axis of the neck. Thus, reduction was effected in nine out of thirteen cases with head-fracture, two, however, died soon after. Among the patients that survived, four good results were obtained—three by 'closed' methods, one by operation.

With fracture of the shaft, eight reductions were obtained out of sixteen cases—seven by 'closed' methods, one by operation. The result after reduction in seven of these patients was good, in one case it is not recorded.

It would appear then that if a patient sustains fracture of the shaft with hip-luxation, or survives the force that breaks the femoral head, a good result may sometimes be got by making early trial of 'closed' reduction. 'Closed' methods are likely to fail when the femoral lever is broken—as in fractures of the neck—by transverse fracture of the femoral head.

Note A On Union of the Fracture before Reduction of the Hip-luxation—Astley Cooper believed that reduction was 'impracticable' apart from union. Actually union had occurred in only five of the twenty-two cases where reduction was obtained, one of these five being treated by operation. Thus seventeen luxations out of the twenty-two were reduced before the associated femoral fracture had united—twelve by 'closed' methods with six good results, five by operation with three good results.

Note B On the Interval between the Accident and Reduction of the Luxation—With head-fractures the only long interval was three months, occurring in a case ending in ankylosis, where the luxation was reduced by operation. In one case treated by 'closed' reduction the interval was three weeks, and here it was thought that the fracture had partially united. Six other luxations were reduced by 'closed' methods with a maximum interval of five days, one of these recurred on the nineteenth day.

Relatively long intervals are found in the only three successful cases with neck-fractures—thirty-eight days and ten weeks in the two cases reduced by 'closed' methods, seven months in the case reduced by operation. In all three the fractured neck had united before reduction was tried.

In two shaft-fractures there were intervals respectively of three weeks before 'open', and of five weeks before 'closed', reductions, while in the remaining four

cases where the interval is mentioned the luxation was reduced by 'closed' methods within five days (Cooper's case, with a five-week interval, was the only one in which the shaft-fracture had united)

In cases where attempts at reduction failed the interval is recorded in only two, one of head-fracture (three months), one of shaft-fracture (five months), in both the femoral head was resected death followed immediately in the first case

PART II

'SURGICAL' FRACTURES OF THE FEMUR CAUSED BY ATTEMPTS AT REDUCING LUXATIONS OF THE HIP

The accident of fracturing the femur during an attempt to reduce a luxation of the hip confronts the surgeon with one or other of the conditions already reviewed in this paper We can therefore consider briefly, under three headings, twenty-one reported cases of a mishap which has severally affected the *head*, the *neck*, and the *shaft* of the femur Of these twenty-one cases, one fracture was of the femoral head, seventeen were of the femoral neck, and three were of the shaft *

I 'SURGICAL' FRACTURE OF THE FEMORAL HEAD WITH HIP-LUXATION

While this paper was under revision, we found what appears to be the only recorded example of this complication, in an article on old traumatic dislocation of the hip Miltner and Wan report the case of a Chinese male, aged 36, whom they saw first on July 1, 1925 Four and a half months previously he had fallen from a wagon, dislocating his right hip An X-ray showed that the head was totally displaced through the posterior part of the obturator foramen A layer of dense material covered most of the surface of the dislocated head, and it appeared to consist of calcified ligamentous and periosteal tissue, it is seen in the X-ray published by these authors to constitute "an imitation of the primary acetabulum" The inferior rim of the true acetabulum was also fractured, but the femoral head seemed intact except for a moderate amount of osteoporosis Nine days after admission 'closed' reduction was performed under ether During the manipulations, which lasted an hour, and were "very severe", a cracking sound was heard, following this the head slipped into the acetabulum An X-ray was at once taken, which showed that the reduction had been obtained at the expense of a fracture through the head "Several broken fragments of the head remained in the obturator foramen" After the trauma of reduction a large hæmatoma appeared in the

* The two cases described respectively by Physick, and Harris and Randolph, and quoted by Hamilton from Gibson, in which femoral fracture occurred during attempted reduction, are not included, since we have been unable to find what part of the femur was broken In Harris and Randolph's case, a boy aged 12, with an unspecified hip-luxation, the bone fractured during extension and counter-extension, at the moment of rotating the leg and drawing it forcibly outward with a towel In Physick's case the fracture was caused in much the same way by force exerted in a lateral direction with an additional pulley We could get no further detail

right groin with a rise of temperature between 38° and 39° C Two weeks later baking, massage, and active and passive movements were begun Two months later the patient walked on his right leg, but still complained of pain He had then 60° of flexion, though motion in other directions was greatly limited One year later the right hip was painful on weight-bearing, but nevertheless was greatly improved when compared with its previous condition

II 'SURGICAL' FRACTURE OF THE FEMORAL NECK WITH HIP-LUXATION

Seventeen instances of this fracture are recorded *

Sex and Age Incidence.—Twelve of the seventeen cases were males, two were females, in three the sex is not stated The age is specified in twelve cases, and one patient is described as "middle-aged" The youngest was 15, the oldest 82, the average age is 46—a higher average than occurs in any other group of femoral fracture with hip-luxation

Interval between the Original Accident and the Attempted Reduction—This interval is recorded in eleven cases, it varies from twenty-four days to one year, the average interval being four months

The Luxation and the Neck-fracture—The *luxation* is specified in eleven cases, in eight it was posterior, including three luxations described as sciatic One obturator luxation is recorded, one pubic, and one anterior and upwards This last, described by Verneuil, was found post mortem four years after the fracture of the femoral neck in a man aged 82, the head lay in the notch between the ilio-pectineal eminence and the anterior inferior spinous process

Precise descriptions of the *fracture* are rare, in three cases it was said to be close to the femoral head, and this was its probable site in the cases where the exact position is not specified

Manipulations which Caused the Neck-fracture.—These are specified in eight cases, and are described respectively as follows (Note except in three cases—specified in brackets—the luxations were all of the *posterior* variety) flexion, flexion with circumduction, abduction, abduction with inward rotation (*anterior and upward luxation*), abduction with manual traction (*obturator luxation*), pulley traction with "slight rotation", external rotation in one case blame is laid on "rotation" alone (In one other case, Lisfranc's *pubic luxation*, the force is merely described as "great")

Treatment and Results of 'Surgical' Fracture of the Femoral Neck—Most of the authors who admit this accident end their record at the point of fracture In only five cases out of seventeen—Thomson's, Albert's, Bryck's, the case quoted by Wippermann, and Wippermann's own case—have we found indications of what happened afterwards Thomson's reduction of an unspecified luxation with 'surgical' neck-fracture has already been fully described in *Part I* with the

* We have included Lisfranc's case with pubic luxation in this group of 'surgical' fractures Lisfranc himself believed that the fracture was due to the original injury, but his own account of the case suggests instead that it was caused by surgical intervention The right lower limb was shortened with the foot everted and the great trochanter raised The thigh was in a position of extension Flexion and inward rotation were impossible Reduction was tried by traction with great force a sudden crack was heard, and signs of fracture of the femoral neck were found

cases due to ordinary trauma, in order to supplement the meagre information regarding their treatment. It is sufficient here to recall that the luxation was reduced with long hooks through anterior and posterior incisions. In no other case where fracture of the femoral neck was caused solely by attempting to reduce a hip-luxation could we find that the luxation had been afterwards reduced,* though in the case quoted by Wippermann the patient is said to have walked well. Albert states that in his case, where the femoral neck was fractured in the attempt to reduce a posterior luxation, the patient, who had previously walked with crutches, now used a stick only. Bryck and Wippermann, describing the end-results of their own cases, mention the only complication that we could find in this group—in both, an abscess formed round the separated proximal fragment and the necrotic head was removed. Wippermann's patient first walked with crutches and a stick, later she was provided with a high-heeled shoe, but she could not use it. Bryck's patient came to his clinic eight months after the accident, the necrosed fragment was removed, the patient died of erysipelas twenty-one days after the operation.

The reader is referred for information on the treatment of these misfortunes to what we have written above concerning the kindred cases due to ordinary trauma (*see pp 217, 218*)

III 'SURGICAL' FRACTURE OF THE FEMORAL SHAFT WITH HIP-LUXATION

We have found only three cases of this rare accident, they are severally recorded by Streissler, Malgaigne, and Wight. Two occurred in males aged 14 and 17, the sex of the third case, aged 53, is not stated. The level of the fracture was different in each—just below the trochanter (Wight), in the upper third of the shaft (Streissler), and in the lower third (Malgaigne), the last being the only instance, besides Fergusson's case (*see Part I*), of shaft-fracture in this third of the femur associated with hip-luxation.

Mamipulations which Caused the Shaft-fracture—In Malgaigne's case of posterior hip-luxation external rotation broke the shaft in its lower third. The same movement caused the upper third fracture in Streissler's case of obturator luxation. Wight's subtrochanteric fracture occurred when the thigh was abducted to reduce a sciatic luxation.

Results—In Streissler's case alone was the luxation, which was obturator, finally reduced (1908). When the broken shaft had united, he operated, using Kocher's incision, eleven weeks after the luxation had occurred and six weeks after the fracture. The acetabulum was cleared of fibrous tissue and the femoral head was levered into place, he then extended the limb, at first in abduction, then in the straight position. The head slipped from the socket while the wound was healing, and was replaced. Following after-treatment by warm baths and passive movement the patient walked well without support two and a half months after operation, the movements of the hip were moderate. Malgaigne and Wight do not record the end-results of their unreduced cases.

* In Tunnecliff's case (*see p 214*) re-fracture of the femoral neck did not interfere with reduction, with which it seems to have synchronized.

These accidents should be treated at once on the lines suggested in *Part I* of this paper (see p 222)

Our thanks are due to Dr R A Gardner, Director of the Radiological Department, Faculty of Medicine, Cairo, for his radiographs, to Miss Gwillim for her skilled after-treatment of *Case I*, and to Drs Mohamed el Zeneiny and Botros Youssif for their assistance in experiments on the cadaver

BIBLIOGRAPHY

- ALBERT, *Lehrbuch der Chirurgie*, 2nd ed, iv, 298
 ALLIS, *Trans Med Soc, Pennsylvania*, 1879, xii, 682
 BAINBRIGGE, *Lond Med Gaz*, 1846, iii, 1004
 BIGELOW, Quoted by Hamilton, loc cit, 1042
 BIRKETT, J, *Trans Med Chir*, London, 1869, liii, 133
 BLOXHAM, *Lond Med Gaz*, 1833, Aug 24
 BORCHARD, *Deut Zeits f Chir*, 1902-3, lxi, 572
 BRAUN, *Arch f klin Chir*, 1891, 109
 BRYANT (quoting Birkett), *Practice of Surgery*, 1872, 1761
 BRYCK, *Arch f klin Chir*, 1873, xv, 279
 BUCHANAN, J J, *Surg Gynecol and Obst*, 1920, xxi, 462
 CHITWOOD, *Med and Surg Reporter*, Philadelphia, 1867, xvii, 353
 CHRISTOPHER, R F, *Arch of Surg*, 1924, xii, 1049
 COOKE, *Lancet*, 1864, i, 37
 COOPER, SIR ASTLEY (reporting Forster's case), *Treatise on Dislocations and Fractures of Joints*, 2nd ed, 1823, 62, 63
 COOPER, SIR ASTLEY, *Dislocations and Fractures*, American ed, 1844, 40
 COTTON, F J, *Dislocations and Joint-fractures*, 2nd ed, 506
 CRILE, G W, *Ann of Surg*, 1891, May, 373
 DAWSON, *The Chmc*, 1874, Oct 17
 DE CHEGOIN, H (quoted by Raillard), "Luxations de la Cuisse compliquees de Fracture du Femur", *These de Paris*, 1866
 DELAGARDE, *St Bart's Hosp Rec*, 1866, ii, 183
 DE MORGAN, *Med Times and Gaz*, London, 1873, i, 549
 DOUGLAS, J, *Edin Monthly Jour Med Sci*, 1843, v, 241
 DREHMANN, *Beitr f klin Chir*, 1896, xvii, 787
 DURAND and DESTOT, *Lyon med*, 1904, cii, 212
 ETEVE, *Gaz Med*, 1838, 757
 FERGUSON, *Practical Surgery*, 1843
 GARTNER, *Wurtemberg, Corresp Blt*, 1872, xli, 36
 GAUB, O, Quoted by Buchanan, loc cit, 471
 GWYNNE, Quoted by A Cooper, *Dislocations and Joint Fractures*, American ed, 1844, 88
 HAASE, 1800, Inaug Disc, Lipsiæ (Leipzig)
 HAMILTON, FR H, *Fractures et Luxations*, French ed, 1884
 HARCOURT, *Buffalo Med and Surg Jour*, 1870-1, v, 241
 HARRIS, G R, *Jour Amer Med Assoc*, 1924, lxxvii, 1608
 HARRIS and RANDOLPH, Quoted by Gibson, *Surgery*, 6th ed, i, 389
 HENRY, A K, *Brit Jour Surg*, 1924, vii, 84
 HEY GROVES, E W, *Ibid*, 1927, xiv, 514
 HINSDALE, G, *Jour Amer Med Assoc*, 1923, lxxv, 459
 HOEFTMANN, *Deut med Woch*, 1904, xxx, 325
 KAMMERER, *N Y Med Jour*, 1889, xli, 175
 KOCH, *Berl klin Woch*, 1882, 492
 KRACKOWIZER, Quoted by Hamilton, loc cit, 1042
 LEVIN, J J, *Brit Jour Surg*, 1923, vi, 388
 LISFRANC, Quoted by Raillard, "Sur quelques Luxations de la Cuisse compliquees de Fracture du Femur", *These de Paris*, 1866
 LOSSEN, *Deutsche Chirurgie*, Lief 65, 55
 MALGAIGNE, *Traite des Fractures et des Luxations*, 1847, ii, 146, 830
 MARKOL, *N Y Jour Med*, 1855, 30
 MILTNER and WAN, *Surg Gynecol and Obst*, 1933, Jan, 79
 MONKS, G H, *Ann of Surg*, 1911, li, 393

- MOXON, *Med Times and Gaz*, 1872, 1, 96
 MURDOCH, *Trans Med Soc, Pennsylvania*, 1878, 144
 NICHIARELLI, C, *Ann Ital di Chir*, 1927, Anno vi, fasc 8, 833
 O'CONNOR, *Provincial Med Jour*, 1844, 77
 PHYSICK, Quoted by Gibson, *Surgery*, 6th ed, 1, 389
 PLATT, H, *Brit Jour Surg*, 1932, vi, 603
 POST, *N Y Med Rec*, 1878, viii, 366
 PRINCE, D, Quoted by Hamilton, loc cit, 1041
 RICHET, *Bull gen de Thérap*, 1853, xlv, 102
 RIEDEL, *Beilage z Zentralb f Chir*, 1885, 92
 ROBERTS, J B, *Ann of Surg*, 1896, xiii, 207
 ROSE and LELLMAN, Quoted by Hamilton, loc cit, 1042
 SATTA, *Chir di Org di Movimento*, 1922, vi, 97
 STIMSON, L A, *Practical Treatise on Fractures and Dislocations*, 6th ed, 1910
 STIMSON, L A, *N Y Med Jour*, 1889, 1, 163
 STRIFSLER, *Beitr z klin Chir*, 1908, lvi, 571
 TANTON, *Fractures du Membre inférieur* (Collection Le Dentu et Delbet, 1913)
 THOMSON, C E, *Jour Amer Med Assoc*, 1904, viii, 884
 THORNHILL, *Lond Med Gaz*, 1836, July
 TRAVERS, *Lond Med and Chir Rev*, 1828, Nov, 239
 TUNNCLIFF, *Amer Jour Med Sci*, 1868, li, 113
 VERNEUIL, *Bull Soc Chir*, 1870, vi, 145
 WIGHT, J S, *Hosp Gaz*, 1879, Sept 13
 WIPPERMANN, *Arch f Klin Chir*, 1885, xxi, 440
 WISEMAN, RICHARD, *Eight Chirurgical Treatises*, 1676, Book vii, Chap 8 London.
 WOOD, J R, Quoted by Hamilton, loc cit, 1041

FRAGILITAS OSSIIUM TARDA

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CONGENITAL brittleness of the bones is a condition which has long been recognized. It is evidenced by multiple fractures from insufficient cause, such cases are usually grouped under the general term 'osteogenesis imperfecta'. As the clinical types vary considerably it is usual to make further sub-divisions. Thus Thomson¹ makes two, and in the first puts cases where the disease appears before or at birth and is usually fatal, while in the second he includes cases where the child, born apparently healthy, shows multiple fractures during early childhood or later.

Knaggs² in 1924 described four main types (1) The foetal—the child is still-born or survives for a short time, with multiple fractures, (2) The infantile—a continuation of (1), the child lives two to three years—a less severe form than the foetal type, (3) The childhood and adolescent type or fragilitas ossium tarda—to be described here, (4) The adult form characterized by extreme fragility of the bones in adult and later life with multiple fractures.

Fairbank³ similarly divided the condition into four clinical types (1) The foetal—similar to Knaggs' No 1, (2) The honeycombed bone type—older children, extreme fragility, large unossified areas in the bone, no definite family history, (3) The slender bone type—similar to Knaggs' No 3 or fragilitas ossium tarda, (4) The marble bone type or Albers-Schonberg disease. The last is undoubtedly a variety of osteogenesis imperfecta, but is often considered as a separate entity because so many cases have now been described. Fairbank has excluded Knaggs' doubtful adult form.

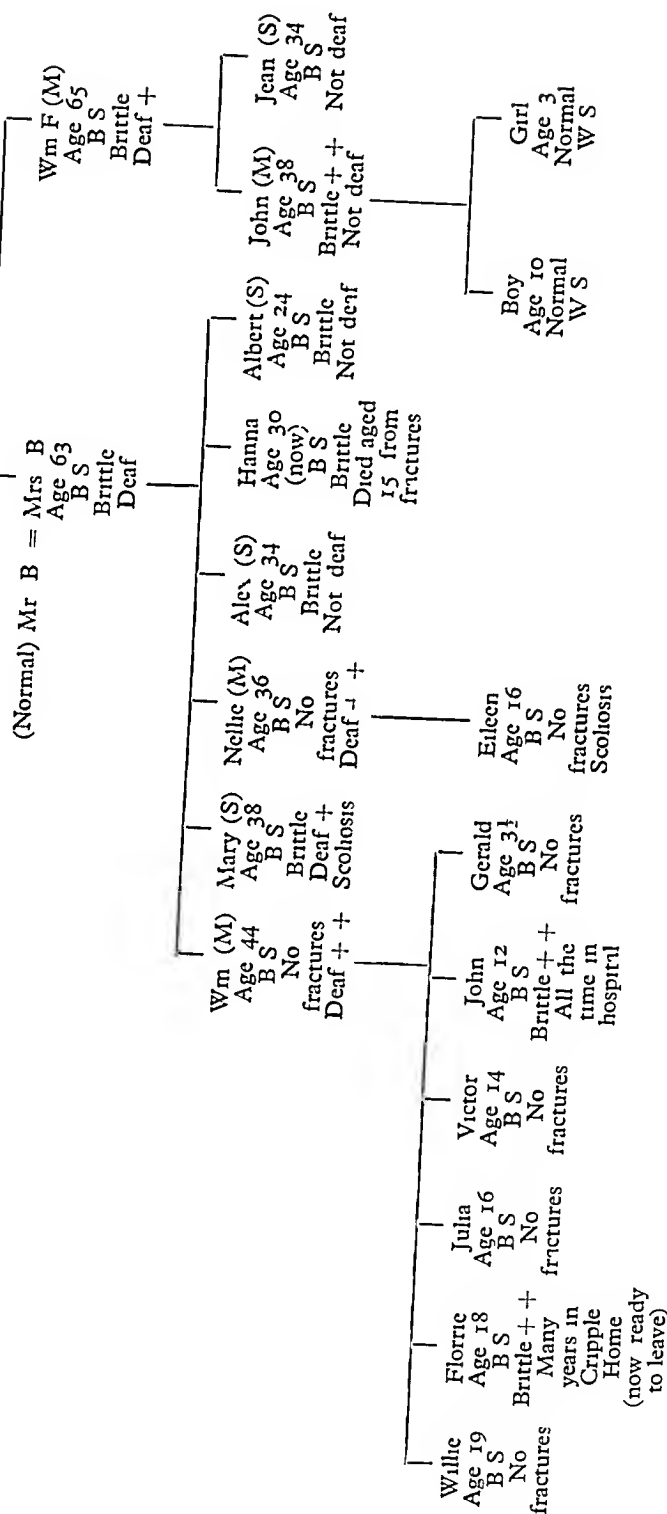
Fragilitas ossium tarda is the only other variety which has yielded clinical material with sufficiently constant symptoms and signs to allow a definite syndrome to be drawn up. For the others it would require a central body to collect, segregate, and classify the individual cases, just as the Codman Registry in America was able to clarify the dense fog which surrounded the bone sarcomata.

The following critical study of a family suffering from fragilitas ossium tarda (see genealogical tree and Fig 153) is given in order that it may be added to the others already on record and so aid in making more definite at least one group of osteogenesis imperfecta.

Fragilitas ossium tarda is an hereditary disease. It is conveyed alike by mother (Case 2) or father (Case 3). The brittleness may even miss a generation (Case 4), but if it does so, the parent always has blue sclerotics. It attacks with equal frequency and with equal severity both sexes. The outstanding sign or symptom is an extreme brittleness of the bones, which break under minimal strains. The most usual bones involved are the long bones of the extremities, but fingers, ribs, clavicles, etc., may be involved. The fractures rarely appear till after 2 to 3 years of age—hence fragilitas tarda (Looser⁴)—and occur only infrequently after 16 to 17, i.e., in the typical case the child has anything from five to twenty breaks during the age

GENEALOGICAL TREE OF FAMILY WITH FRAGILITAS OSSIUM TARDA

(Unknown) Mrs F = Mr F (family all known—normal)



B S = Blue Sclera
 M = Married
 W S = White Sclera
 S = Single

period 3 to 17 There is rarely the very large number of fractures found in the foetal and infantile variety of osteogenesis imperfecta

Accompanying the brittleness of the bones there is in addition a dull blue discoloration of the sclerotics (*Fig 154*), most marked towards the iris This is a more dominant factor than brittleness, so that in a family anything up to 90 or

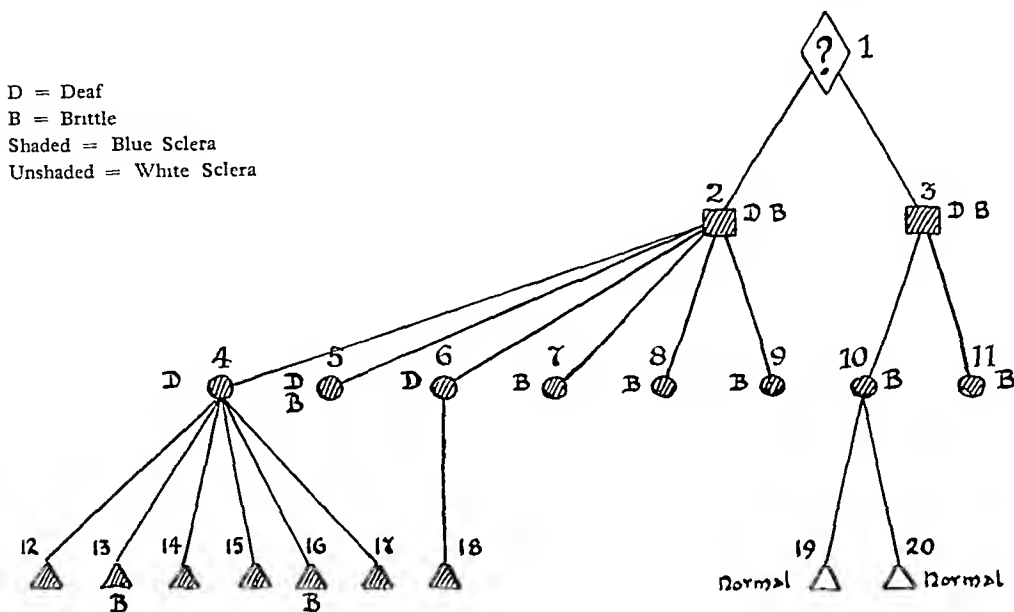


FIG 153—Diagrammatic representation of genealogical tree, with cases numbered

100 per cent may have the blue sclerotics, but roughly only 50 per cent may be expected to show fractures In such a family no case of brittle bones occurs with a white sclera

A further item in the syndrome is the deafness This occurs in approximately 25 per cent of those with blue sclerae It attacks equally those with brittle bones



FIG 154—To show the blue sclera often described as 'Wedgwood blue'

(Cases 2 and 3) and those who have shown no fractures (Cases 4 and 6) The interesting fact is that the deafness only appears after the brittleness subsides Deafness is rarely present until after twenty years of age All who recover from fragility of bones strongly emphasize how very hard their bones become, and usually

record with pride the severe accidents from which they have come out unscathed—accidents which, in earlier life, would certainly have broken many bones. This hardening, late in appearing, should possibly be connected with the deafness, which is generally agreed to be of the otosclerotic type.

A tendency to sprain readily is recorded by many writers, and several patients in my series spontaneously gave this information, possibly this is a further proof of a fault in the quality or quantity of the body fibrous tissue, as is evidenced already by the transparency of the sclera. Two members (*Cases 5 and 18*) had a scoliosis of the postural type—also explained by some writers as a further sign of the faulty character of the fibrous tissue.

A well-marked arcus senilis was present in several of the cases. This started to appear with the other sclerosing changes, and was present in a patient as young as 30 years. An early arcus senilis is in some families an hereditary characteristic and of no significance, but the marked frequency with which it is found in cases of fragilitas ossium tarda must be more than a mere coincidence.

The height of this family of four generations was average or rather shorter, but the members with brittle bones were decidedly smaller and some definitely undersized. By some this is claimed to be due to the multiplicity of the fractures, with naturally subsequent shortening due to impaction, absorption, or overlapping. This is obviously a fallacy, as it is an interesting fact that often the same limb is continually broken, whilst the other escapes (Stewart²), so that unilateral shortening is what one would expect. This occurred in one case (Woodside³), and two inches were removed from the sound leg to straighten the pelvis.

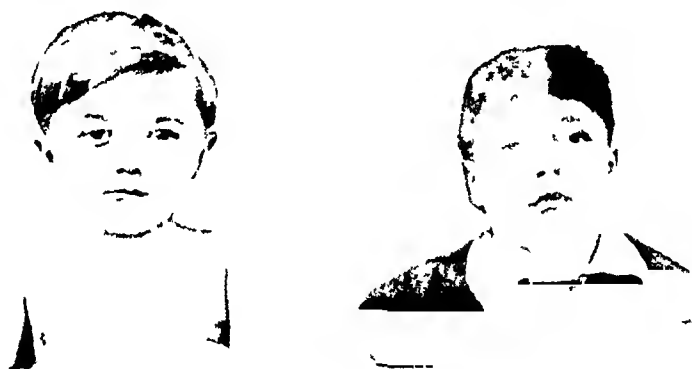


FIG 155—*Case 16*. To illustrate the shape of the head and the tilt of the ears. On the right is a boy of the same age for comparison.

A careful examination of the epiphysis shows that early synostosis takes place with cessation of growth. This would appear to start at 15 years and onwards, so that *Case 13* (fragile) was really further advanced in ossification than *Case 12*, her brother older by two years and non-fragile.

The shape of the skull is a sufficiently constant sign to receive consideration (*Fig 155*). The appearance from the front is that of an enormous calvarium—almost hydrocephalic—and a very broad forehead with the parietals tending to bulge over the ears, in contrast the lower part of the face and chin are so very small that it

gives the face a triangular appearance (with the apex below) From above, however, the skull is seen to be flat, and thus probably its actual capacity is in no way greater than normal This is usually explained by the pressure produced by a semi-diffuent brain upon side walls less resistant than usual This is probably correct, as it is a more marked feature of the foetal and infantile varieties of osteogenesis imperfecta, where it is admitted that the fragility is greater and where the skull may even be almost of the consistency of a membranous bag

Biochemical examination of the blood was carried out upon several members in this series by Dr J A McVicker⁷ In these and other cases of fragilitas ossium tarda he found the blood calcium, blood phosphorus, and phosphatase all within the normal limits This is in accordance with the findings of other workers, and is further confirmed by the fact that no improvement appears to occur in these cases with calcium, phosphorus, parathyroid, or vitamin therapy

INDIVIDUAL FEATURES

The Scleræ—The blueness is deep and clear in the child, but gets greyer and duller in old age, although it never disappears as does the tendency to fracture (see Fig 154) This is a well-known familial characteristic Myles Bickerton⁸ states that since 1903 there have been some 463 defective individuals recorded Of the adults with blue sclerotics 60 per cent had liability to fractured bones, 60 per cent had otosclerosis, and 44 per cent had all three defects Following the Mendelian Law it is thus a dominant characteristic (Groenow⁹) Its pathology is variously explained Bickerton says that the sclera is too thin and transparent Knaggs contends that there is no diminution in the thickness of the sclera and no difference in its microscopic structure, and therefore he holds that the translucency depends upon some exceptional peculiarity of the fibrous tissue of which it is composed Against this is the fact that Buchanan¹⁰ in an enucleated eyeball found the sclera about one-third of the normal A further suggestion made by Fridenberg (Alexander²⁵) was that the transparency was due to a lack of calcium—a suitable hypothesis and one intended to fit in with the brittleness of the bones and their decalcified appearance The hereditary tendency of blue sclerotics has been frequently pointed out Peters¹¹ in 1908 described a family of eleven members with ten showing blue sclerotics In 1913 he showed the connection with brittle bones Deighton,¹² Cockayne,¹³ Stephenson,¹⁴ Harman,¹⁵ et al, in interesting pedigrees, have pointed out the dominant hereditary factor that the blue sclera can be In some of these only was the fragility of the bones mentioned, although it was as early as 1900 that Alfred Eddowes¹⁶ in a very short note to the *British Medical Journal* first pointed out a connection between the two

Many excellent pedigrees showing the relationship between the two conditions have appeared, those of Olaf Blegvad and Holger Haxthausen,¹⁷ Burrows,¹⁸ Van der Hoeve and de Klyn,¹⁹ Freytag,²⁰ Bronson,²¹ Voorhoeve,²² Willard,²³ Simmonds,²⁴ Alexander,²⁵ Stewart,⁵ to mention a few, are typical

The Bones—Radiologically in the child in the active period what appears most striking is the extremely slender shaft which stands out in contrast with the expanded extremities This is most noticeable in the long bones of the limbs The shaft, although slender, rarely bends, angulation if present is usually due to an old fracture (Fig 156) The cortex is said to be thin, but rather, I think, a cortex

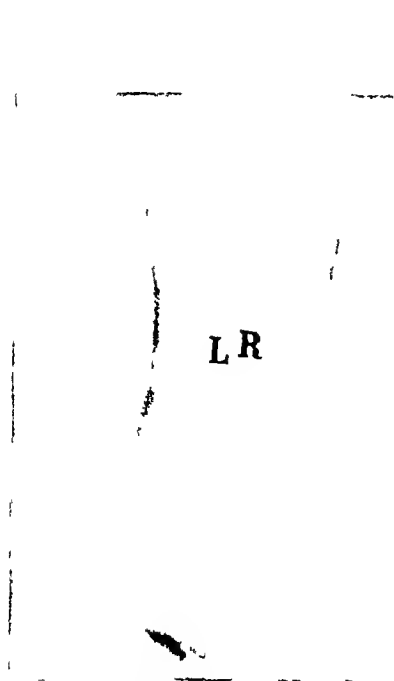


FIG 156 —To illustrate the decalcified bones, which give a poor contrast with the soft parts



FIG 157 —Knee joint of case of osteogenesis imperfecta of infantile type for comparison



FIG 158 —Types of fracture and the structure of the bone. It appears almost cystic in parts

with little underlying cancellous tissue and enormous medullary cavity is the rule. In the extremities the reverse holds good. There is a wide-webbed honeycomb of cancellous tissue with a thin limiting cortex. The lamellæ in the honeycomb lack the uniformity and regularity of normal bone. As a result of the thin cortex the opposing surfaces are often flattened in weight-bearing, thus giving a joint the appearance of a horizontal slit, e.g., the knee. The appearance of the bone has a direct bearing upon the type of fracture produced. In the *shaft* it is usually, even in the young, a clean transverse fracture. In the *extremities* it is said that a greenstick fracture is common, a better term would be a 'compression' or 'cancellous' fracture. One cortex is broken, but there is considerable impaction as well (*Fig 158*).

In the cured patient the radiograms are disappointingly normal—the cortex may be denser and thicker than normal, the flat weight-bearing surfaces remain, and there may be the distortion from the old fractures. Beyond this there is little of interest. In the child clean-cut pictures are difficult to obtain, as the bones are less dense than normal and so contrast poorly with the surrounding soft parts, thus the X-rays in this article inadequately demonstrate the condition, although great care was taken in getting a negative with as much contrast as possible.

It is frequently stressed (Stewart⁵) that the fractures appear to favour one limb or one side. This is readily understood by noticing how often the disease, although it is a generalized one, is more marked upon one side than another (*Fig 159*).

A radiogram of the skull shows the parietal bulge (*Fig 160*), but in my series there was no other marked abnormality, although Knaggs affirms that the presence of multiple wormian bones is almost a constant feature.

Biopsy was performed twice upon one of these patients, and both times the most noticeable feature was the extreme density and hardness of the bone (Irwin³⁰). It felt and cut like marble.

This is contrary to expectation and certainly contrary to what the radiogram would suggest. However, there must be some connection with marble bones, or the Albers-Schonberg syndrome. In the latter the hereditary tendency is a strong one, and in some cases blue sclerotics have been described.

Professor Young,²⁹ reporting upon a piece of bone removed in *Case 13*, states —

C 5549 —The general architecture of the bones shows no gross abnormality apart from some increase of density.

The Haversian canals have been encroached upon by a deposition of bone upon their walls. This new-formed bone is rather indistinctly lamellated, contrasting in this respect with the pre-formed bone of the Haversian system, and it would appear to have been deposited in successive stages because it is frequently composed of two or more layers separated by a denser line of cement substance.

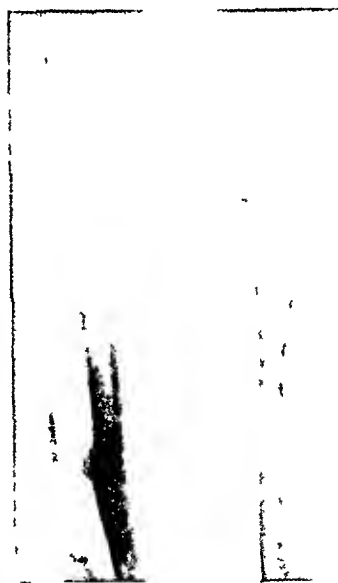


FIG 159 —*Case 13*. Showing difference in the two legs. The right is much thinner and had ten breaks in all. The left is thicker (cf fibula). The callus is the result of operation.

These changes can be readily correlated with the increased consistency of the bone, which was such a notable feature clinically. The increased brittleness is less easily explained, but it may be associated with the imperfect lamellation which has been noted in the bone with the Haversian canals.

Deafness—Deafness as part of the syndrome was first pointed out in 1918 by Van der Hoeve and de Klyn¹⁹, who described two families. In the first, of 22 members, 10 had blue scleræ, 10 had brittle bones, and all were deaf. In the second, of 6 members, 6 had blue scleræ, 5 had brittle bones, and all were deaf. In Olaf Blegvad's pedigree,¹⁷ of the 21 members in three generations, 9 had blue scleræ, 8 had fractures, and 5 had deafness. In addition zonular cataract was a familial disease in this family. It is thus seen that the proportion who show deafness or brittleness in any such family is variable.



FIG. 160—Illustrating the shape of the skull. Case 13 on the left, Case 16 on the right. In the centre is a control case for comparison.

It is natural that the connection between brittle bones and deafness was late in being recognized, as the two do not appear concurrently. When the child is attending the surgeon for repeated fractures deafness has not yet appeared, and later, when as an adult he is being treated for deafness, the brittleness of his earlier days is forgotten or of apparently little connection with his present complaint.

Voorhoeve²² suggested that the underlying cause of the deafness was labyrinthine disease, but Bronson,²¹ Crocco,²⁶ and others all hold that it is due to otosclerosis. The latter is well recognized by otologists to be a hereditary disease. Stenvers²⁷ found by radiography that the labyrinthine region was covered with a thick calcareous substance and suggested that deafness was due to a reduction of the nutrition of the part.

Whatever the cause, clinically the deafness comes on insidiously and is not amenable to any treatment. It starts to appear from 20 years and onwards, when the bone fragility has disappeared and when the sclerosis has set in. It is of interest that of the six members of the third generation of my series all had blue sclerotics,

four had fractures, and it is the two who had no fractures who are now deaf. One of those deaf but with no fractures is the father of six children, two of whom were probably the most crippled with fractures in the whole series. The percentage of deafness quoted by any writer is always open to an obvious fallacy in that usually, as in the family here, the last generation has not yet reached the otosclerotic age.

A most striking feature is the return to normality, from being almost crippled, which occurs in these people after a certain age. One man (*Case 7*), a lorry driver, can crank the stiffest engine with an arm that is tortuous and bent as the result of some ten fractures when young. The others similarly are leading strenuous lives. This does not always happen, as in some families the early deformities may have been so great that the patient is left a cripple in adult life.

Cases 2 and 3 are hale and hearty, aged 63 and 65 years respectively, thus the disease does not appear to affect the ultimate expectation of life, although Knaggs² states that as a rule such people are not long-lived.

This possibly arises from the fact that one writer mentions high blood-pressure as occurring frequently in these cases—perhaps another proof of the appearance of premature sclerosis.² An estimation of the blood-pressure was not carried out in my own series.

SUMMARY

1 A detailed pedigree is given of a family of twenty members of four generations. Of these, 17 had deep blue sclerotics, 10 had multiple fractures, and 5 were deaf. In addition several members had scoliosis, a tendency to sprain readily, and an arcus senilis at a comparatively early age.

2 Such a syndrome is typical of the condition of fragilitas ossium tarda, one of the best known and most constant forms of osteogenesis imperfecta.

3 There is an active period of from 2 to 17 years when fractures occur. Union is normal in time and appearance. This is followed at puberty by a second period of extreme sclerosis. This affects bones, making them exceedingly strong, and the ears, producing deafness of the otosclerotic type and (?) an arcus senilis.

4 After the onset of the second period the patient recovers to enjoy a normal life and reach an average old age.

5 Other features are the broad skull and a normal blood chemistry.

6 The questions still to be settled are (a) Why do the fractures delay in appearing till 2 years of age, approximately? Is it due to the persistence of some of the mothers' hormones? (b) Why at puberty does this fragility disappear? Is it due to the appearance of some internal secretion, e.g., ovarian or testicular?

I wish first to record my thanks to all the members of the family here reported. Their interest and help made what might have been a difficult task a pleasure. The willingness with which, even though at inconvenient times, they allowed themselves to be examined and radiographed—from grandmother of 63 to grandchild of 4—is hard to realize. I wish to thank Mr H P Hall for access to *Case 16*, and Mr Woodside for access to and information regarding *Case 13*, also Professor Young for valuable assistance regarding the pathology of affected bone removed, and Dr Maitland Beath for help in the interpreting of the radiograms.

REFERENCES

- ¹ THOMSON, *Cluneal Study and Diseases of Sidel Children*, 1921, Oliver & Boyd
- ² KNAGGS, R L, *Brit Jour Surg*, 1924, vi, 757
- ³ FAIRBANK, H A T, *Ibid*, 1927, July, 120
- ⁴ LOOSER, *Grenzgeb der Med*, 1906, xvi, 165
- ⁵ HALL-STEWART, *Brit Med Jour*, 1922, Sept 16
- ⁶ WOODSIDE, C J A, Personal communication
- ⁷ MCVICKER, J A, Personal communication
- ⁸ MYLES BICKERTON, *Brit Med Jour*, 1934, Jan 20
- ⁹ GROENOW, *Graffe-Sænnsh Hau Buch*, 1920
- ¹⁰ BUCHANAN, *Trans Ophthalmol Soc*, 1903, xliii, 267
- ¹¹ PETERS, *Klin Monats f Augenheilk*, 1908, 130, 1913, 594
- ¹² DEIGHTON, *Ophthalmoscope*, 1912, 188
- ¹³ COCKAYNE, *Ibid*, 1914, 271
- ¹⁴ STEPHENSON, *Ibid*, 1915, 278
- ¹⁵ HARMAN, *Ibid*, 1910, 559
- ¹⁶ EDDOWES, *Brit Med Jour*, 1900, ii, 222
- ¹⁷ OLAF BLEGVAD and HOLGER HAVTHAUSEN, *Ibid*, 1921, ii, 1071
- ¹⁸ BURROWS, *Ibid*, 1911, ii, 16
- ¹⁹ VAN DER HOEVE and DE KLYN, *Arch f Ophthalmol*, 1918, xcvi, 81
- ²⁰ FREYTAG, *Klin Monats f Augenheilk*, 1913, ii, 28
- ²¹ BRONSON, *Edin Med Jour*, 1917-8, 240
- ²² VOORHOEVE, *Lancet*, 1918, ii, 740
- ²³ WILLARD, *Med New Philadelphia*, 1887, ii, 734
- ²⁴ SIMMONDS, *Ann of Surg*, xlii, 186
- ²⁵ ALEXANDER, *Brit Med Jour*, 1922, April 29
- ²⁶ CROCCO, *Comm of Hospital Oph*, Buenos Aires, 1920, ii, 38
- ²⁷ STENVERS, see No 19
- ²⁸ HALL, H P, Personal communication
- ²⁹ YOUNG, J C, Personal communication
- ³⁰ IRWIN, S T, Personal communication

CYSTS IN THE REGION OF THE PANCREAS: WITH NOTES OF A CASE

By A E WEBB-JOHNSON

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CYSTIC tumours in the region of the pancreas are among the least common of all abdominal cysts Hale-White,¹ in a series of 6000 autopsies, found only 4 cases, though in a recent publication Mahorner and Mattson² were able to report on 88 patients with pancreatic cysts treated surgically at the Mayo Clinic

Different authors have adopted various methods of classifying these cysts, a comprehensive one being that of Mahorner and Mattson

I *Cysts Resulting from Defective Development—*

- 1 Cysts in infants
- 2 Cysts associated with polycystic disease of the kidney
- 3 Dermoid cysts
- 4 Inclusion cysts

II *Cysts Resulting from Trauma*

III *Retention Cysts*

IV *Neoplastic Cysts—*

- 1 Cystadenoma
- 2 Cystadenocarcinoma
- 3 Teratomatous cysts

V *Cysts Resulting from Parasites*

This classification does not include the possibility of an inflammatory origin for some of the false pancreatic cysts, nor is there any mention of that cystic condition of the pancreas associated with multiple hæmorrhagic tumours of the spinal cord described by Lindau³ Any of the retroperitoneal cysts which were classified by Handfield-Jones⁴ may also occur in this region

The cyst most commonly found would appear to be the pseudocyst or hæmorrhagic cyst, devoid of any epithelial lining and believed to result from trauma

Cystadenomata are extremely rare, being found in only 2 out of 88 cases operated upon at the Mayo Clinic The pathology of these cysts, in which the cyst is formed by proliferation of epithelium, is a matter for conjecture

The following case is noteworthy, not only for the rarity of the condition and the size of the cyst, but also on account of its histology, which differs in one important feature from the cysts of this region which have been previously described A careful study of the histology may throw light on the origin of cysts in the region of the pancreas and help to a clear understanding of their pathology

CASE REPORT

HISTORY—The patient, a woman of 52, had noticed for the last few months that her abdomen was increasing in size. She complained of "slight discomfort and a little shortness of breath."

ON EXAMINATION—The abdominal wall was found to be tightly stretched over a large swelling filling the upper part of the abdomen, being slightly more to the left of the mid-line. The swelling was fluctuant and immovable.

OPERATION—Laparotomy in June, 1930, showed the stomach stretched like a strap across the left side of the cyst and displaced into the left hypochondrium. On dividing the gastrocolic omentum the pancreas was found lying across the anterior aspect of the cyst. The



FIG 161—Macroscopic appearance of the cyst

pancreas was easily separated, but a number of adhesions containing venous channels were present between the cyst and the spleen. The cyst was removed together with the spleen. The liver, kidneys, and pelvic organs were examined and found to be normal.

SUBSEQUENT PROGRESS—Convalescence was uneventful, and the patient remains well four years after operation, except for "a little indigestion and heartburn."

PATHOLOGY—A thin-walled cyst, a foot in diameter (Fig 161). The cut surface shows numerous loculi containing mucoid material. A section of the wall shows a lining of tall columnar epithelium similar to that of a multilocular ovarian cyst. The septa and cyst wall are formed mainly of fibrous tissue but show traces of somewhat degenerate, but none the less definite, muscle fibres. These fibres are large. Some show peripheral nuclei, but no

striations are visible. In some areas they are placed immediately beneath the epithelium. It is not possible to state definitely the nature of the muscle (*Fig 162*)

Examinations of the fluid contained in the cyst showed no trace of pancreatic enzymes

DISCUSSION

The description of this cyst closely resembles those described by Stewart⁵ and by Carling and Hicks⁶. With regard to the etiology of such cysts there are two views which deserve consideration —

1 That these Cysts are True Cystadenomata of the Pancreas — There appears to be no doubt that innocent epithelial tumours of the pancreas do occur, although they are extremely rare. Adenomatous hyperplasia of the islets of

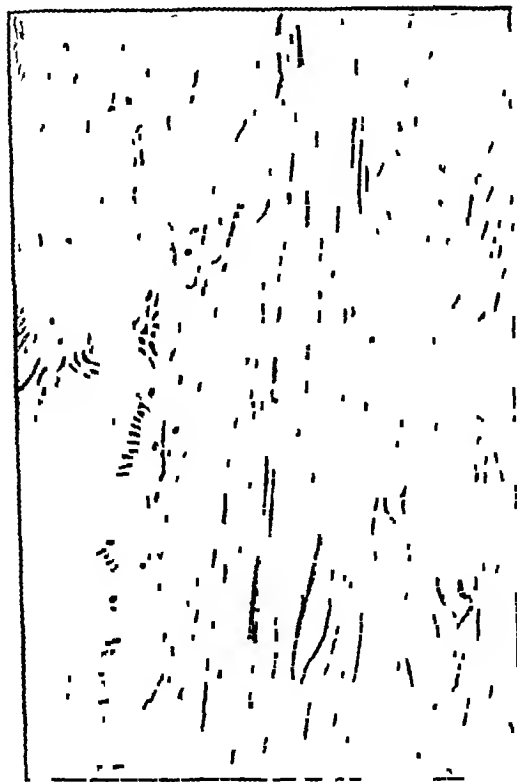


FIG 162 —Section of septa in cyst showing muscle fibres ($\times 185$)

Langerhans has been described, associated with hypoglycæmia. Kaufman⁷ has described what appears to be a true cystadenoma of the pancreas, but the cyst was situated in the centre of the pancreas and was of small size. One of the two cases reported by Mahorner and Mattson is considered by these authors to be a true cystadenoma of the pancreas as histological examination of the cyst wall revealed a "cellular structure not unlike the normal pancreas". As Handfield-Jones states, no cyst in the region of the pancreas should be regarded as of pancreatic origin unless pancreatic tissue is present in its wall or its pedicle.

While true cystadenomata of the pancreas have been found, this explanation of the etiology cannot be applied to the majority of the epithelial cysts found in

this neighbourhood Their lining epithelium bears no resemblance to that of the pancreas, they occur in contact with the pancreas rather than in it, they closely resemble ovarian cysts, and similar cysts have been found in the retroperitoneal tissues in all parts of the abdomen between the inferior surface of the liver and the region of the broad ligament

2 The Theory of Developmental Error—The view that these cysts may result from malposition or sequestration of certain cells in early embryonic life is an interesting one which appears to find general acceptance at the present time

Having regard to the fact that they possess a definite secretory epithelium there would appear to be several possibilities

a That they are Derived from 'Strayed' Genital Cells—Felix⁸ believes that genital cells are not derived from the specially differentiated coelomic cells but from the segmentation cells at an earlier period of foetal life They travel from the region of the cloaca into the root of the mesentery and thence into the urogenital fold It is possible that some of these cells may fail to reach the urogenital fold and become a source of teratomata

While this theory is acceptable for rare cases of retroperitoneal and peritoneal dermoids, such as that previously described by one of us,⁹ it is difficult to apply it to other cysts unless they can be shown to contain derivatives of all three germinal layers

It is of interest to note that Ewing,¹⁰ following Nicholson's¹¹ views, previously discussed by one of us¹² in regard to embryoma of the testis, believes that all testicular tumours, whether epithelial or connective-tissue in nature, are true teratomata, the predominant tissue representing a 'one-sided' development of one of the three germinal layers, the other two being in abeyance Macleod,¹³ in a description of struma ovarii, points out that it is generally accepted at the present time that the thyroid tissue is part of a teratoma and has grown out of proportion to the other structures

If this 'one-sided' development of a teratoma is accepted in other regions, there appears to be no reason why it may not apply to these epithelial retroperitoneal cysts

b That they are of Urogenital Origin—This theory only differs from the last in that the suggested maldevelopment or 'separation' takes place at a later stage in embryonic life Zuckerkandl¹⁴ has demonstrated the persistence of mesonephric tubules long after the normal date of their disappearance, and obviously any one of these may be the starting-point of a cyst

c That they are of Enterogenous Origin—Cysts derived from sequestration of part of the gut have been described in a number of cases Usually unilocular, sometimes multiple, they are lined by columnar epithelium and have traces of plain muscle in the wall The majority have been found at the ileocaecal angle and in the mesentery At the junction of the fore- and midgut the pancreas and liver are developed by a process of budding Abnormal budding can take place, as is shown by the occurrence of congenital duodenal diverticula, and it is therefore possible that some of the epithelial cysts in this region may be derived from the gut

Of these three views the one suggesting a Wolffian origin is generally accepted It has the advantage that it serves as an explanation for those cysts similar to the one described in this case but found in the retroperitoneal tissues in the iliac fossæ, and in the broad ligament

Amongst gynaecologists the view of a Wolffian origin has not found favour when applied to a multilocular pseudomucinous cyst arising in an ovary Ewing¹⁵ states that cysts of Wolffian origin may arise in the ovary but they are small and situated in the hilum Wilfred Shaw¹⁶ recorded 23 cases of ovarian dermoids in 9 of which there was a combined pseudomucinous cystadenoma and dermoid cyst—an incidence of 39·1 per cent Dermoid cyst and cystadenoma formed part of an original tumour with no line of separation He believes that one area of a dermoid cyst turns into a cystadenoma, a view which is in support of a teratomatous origin

We have been unable to find any recorded case of a retroperitoneal cyst in this region containing muscle fibres Their presence in the wall and septa of the cyst in this case is a definite indication of a foetal or teratomatous origin

If the muscle is unstriated, its presence and its close association to the lining epithelium is in favour of an enterogenous origin, a sequestration of foetal gut If it is degenerated striated muscle, the cyst must be regarded as a teratoma

The fact that the veins from the cyst joined the splenic vein, part of the portal system, is not decisive evidence in favour of an enterogenous origin, since the spleen is developed around vessels in the dorsal part of the ventral mesentery, and, if we accept Felix's views, there is nothing to prevent a 'strayed' genital cell giving rise to a teratoma in this situation

REFERENCES

- ¹ HALF-WHITE, *Guy's Hosp Rep*, 1900, liv, 17
- ² MAHORNER and MATTSON, *Arch of Surg*, xlii, 1019
- ³ LINDAU, *Proc Roy Soc Med*, 1930, xlii, 363
- ⁴ HANDFIELD-JONES, *Brit Jour Surg*, 1924, vii, 119
- ⁵ STEWART, *Edin Med Jour*, 1926, xxxiii, 432
- ⁶ CARLING and HICKS, *Brit Jour Surg*, 1925, xiii, 238
- ⁷ KAUFMAN, *Pathology*, 11, 1044 (trans by Reimaur Lewis)
- ⁸ FELIX, Kiebel and Mall's *Human Embryology*, 11, 882
- ⁹ WEBB-JOHNSON, *Brit Med Jour*, 1925, March 28
- ¹⁰ EWING, *Neoplastic Diseases*, 840
- ¹¹ NICHOLSON, *Guy's Hosp Rep*, 1907, lxi, 249
- ¹² WEBB-JOHNSON, *Brit Med Jour*, 1925, Dec 5
- ¹³ MACLEOD, *Proc Roy Soc Med*, 1932, xlv, 1386
- ¹⁴ ZUCKERKANDL, Quoted by Handfield-Jones
- ¹⁵ EWING, *Neoplastic Diseases*, 652
- ¹⁶ SHAW, WILFRED, *Jour Obst and Gynaecol*, 1932, xliix, 234

GANGRENE FOLLOWING FRACTURES (EXCLUDING GAS GANGRENE)

BY HAROLD DODD, LONDON

ALTHOUGH innumerable fractures are continually occurring with all degrees of trauma, fortunately gangrene, apart from infection by gas gangrene and other virulent organisms, rarely follows a fracture. I have searched the records, and since the War have found only ten cases published, these are epitomized here. Between the years 1850 and 1914, thirty cases were reported. They are tabulated later and the more interesting are described. Most of the writing is the work of German surgeons.

"The complication of gangrene is an uncommon one if I am to judge from the meagre reference to it to be found in the literature at my command." Thus Ormsby¹ prefaced an article in 1911, when he published two cases.

Investigation of the reports of the London teaching hospitals during the years 1920-30 did not reveal a case, although there must have been a few, they were probably considered unworthy of mention.

Gregora,² who has studied the condition, found 4 instances of gangrene in 606 fractures. He stresses that although these cases are rare, they are important on account of (1) The difficulty of diagnosis, (2) The steps to be taken to prevent gangrene and so save the limb.

This communication describes a patient of Mr. Watson Jones and two of my own. I have extracted the relevant cases from the literature, excluding the numerous ones of gas-producing and other infections. I have omitted the War reports of gunshot wounds complicating bone and vessels, they were exhaustively dealt with by the late Sir George Makins.³ The present work is confined to the accidents of civil life. From its study I have collected points for the diagnosis of gangrene threatening to complicate a fracture, and to avert its onset various surgical procedures are described.

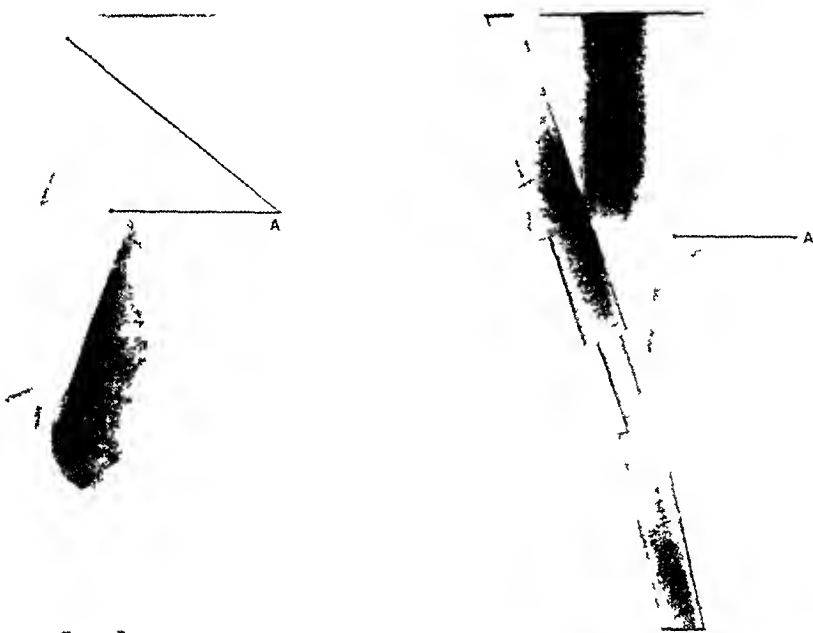
PERSONAL CASES

Case 1—On June 16, 1932, J. B., aged 59, a stableman, was kicked by a horse. His left femur was fractured at the junction of the middle and lower thirds (*Figs 163, 164*). Over this, on the antero-internal surface of the thigh, was a subcutaneous, freely oozing laceration 1 in. long. The limb was immediately manipulated and mounted in a Thomas knee-splint.

I saw the patient the next day, when the leg was dead white, cold, painless and anæsthetic below the knee. No pulsation was detected in the popliteal, posterior tibial, or dorsalis pedis arteries. A diagnosis of thrombosis and occlusion of the femoral artery at the site of the fracture (approximately at the lower end of Hunter's canal) was made.

The X-ray showed that the fragments were in poor alignment (*Figs 163, 164*), and thus it was considered that they might be interfering with the lumen of the vessel. A pin was inserted through the os calcis, and a satisfactory position was obtained by extension. After this the level of warmth crept a little farther down towards the middle of the front of the leg.

Observation continued for a month, and then mummified dry gangrene had occurred with a line of demarcation passing round the junction of the lower and middle thirds on the anterior surface of the leg, and at the upper and middle thirds on the posterior surface (Fig 165) Thus the area of distribution of the posterior tibial artery was affected more than that of the anterior tibial



FIGS 163, 164—Case 1 Radiograms of fractured lower third of the femur A indicates the calcified femoral artery

On July 14—i.e., thirty days after the accident—I amputated the thigh at the level of the fracture and trimmed the end of the bone. Union had progressed to the extent of a mass of gritty callus. On cutting the femoral artery with a scalpel, its wall was felt to be calcareous, but no plaques were visible to the naked eye, the radiograms (Figs 163, 164) show that the femoral and tibial arteries are calcified. The lumen was clear, the intima was smooth and apparently unbroken—there was no change in its colour or sheen. On dissecting the bifurcation of the popliteal artery, it was found to be blocked by two elongated blackish thrombi. They were quite loose in the artery and easily ‘popped out’. The lower thrombus was $\frac{2}{3}$ in long, bent astride the V of the division, and occluding the posterior branch more than the anterior tibial. The upper piece was 1 in long, it was lying longitudinally in the popliteal artery. The striking feature of these thrombi was that, after being in the vessel for a month, they were still unattached to the wall and quite free in the lumen. The only sign of reaction in the intima was a slight blushing just above the fork, $\frac{1}{8}$ in in diameter.

There was apparently no adherence or attempt at organization in the clots.

The case suggests that the thrombi arose at the time of the accident from a contusion of the femoral artery at the level of the fracture, although all signs of it had disappeared a month later. Further, it demonstrates that a thrombus may exist in a diseased artery for a long period without becoming organized or fixed, and that, should it move, it would cause a spontaneous, unexplainable embolism long after the incident which had given rise to it had been forgotten.

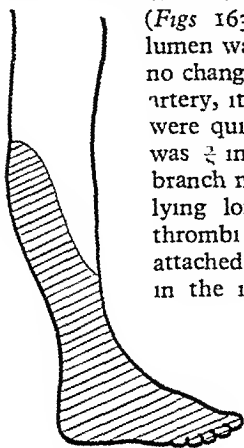


FIG 165—Case 1 Shows the line of demarcation of dry gangrene, following embolism of the termination of the popliteal artery

The patient recovered and is now walking well with an artificial leg. Recently he had a large scrotal hernia which, cured by operation, had been irritated by the buckle of his new limb.

When the gangrene occurred I considered the possibility of a blocked popliteal artery, but I was unable to detect any tenderness over it. However, on dissecting the popliteal artery in the amputated leg, I realized from its deep position that tenderness on palpation would be very unlikely. I was surprised at the length of the wound and the amount of muscle-splitting necessary to expose the region of the bifurcation. In life, the delicate operation of embolectomy in this inaccessible situation would be very difficult, but it would nevertheless be well worth attempting, in view of the grim alternative—inevitable gangrene and amputation.

I have observed sensitiveness over an artery precisely at the site of impaction of an embolus in it, on two previous occasions. On the first it located an embolus at the bifurcation of the common femoral into the superficial femoral and profunda femoris, and on the second it occurred with the brachial artery at the point of origin of its superior profunda branch.

Case 2—On Nov. 8, 1932, W. H., aged 29, was admitted with a comminuted fracture of the middle of the right tibia and fibula (Figs 166, 167). After reduction under an anæsthetic the leg was encased in plaster from the mid-thigh to the interdigital clefts of the toes.



FIGS 166, 167—*Case 2* Radiograms of fractured tibia and fibula

The patient was comfortable all night, but next day his toes were swollen and discoloured. This was not relieved by elevating the leg and trimming the plaster, so the latter was removed the same evening. The site of the fracture appeared to be satisfactory, it was not unduly swollen and there was no unusual discoloration. On the next day the colour of the toes had improved, the limb had been mounted on a back-splint with side-pieces during the night. A plaster was again applied.

In twenty-four hours the toes were again swollen deeply cyanosed, and large blebs appeared on them. The plaster was trimmed but without improvement, and it was finally removed on Nov. 13. The lower half of the limb was dusky and swollen and the large blebs further increased in size. The leg was retained between sand-bags and the foot of the bed was raised on blocks.

By Nov. 15 it was obvious that wet gangrene was present in the foot and lower leg. Gradually the skin fell away, and by Nov. 22 the skin, subcutaneous tissue, and muscles separated from the lower half of the front of the tibia, exposing the bone. A similar process took place behind at the ankle-joint.

On Nov. 29 amputation was performed a hand's width below the tubercle of the tibia. The muscles were greyish-pink and gelatinous, there was only a small amount of bleeding from the peroneal artery and subcutaneous vessels, the anterior and posterior tibial arteries were both thrombosed with a small peri-arterial hæmatoma at the level of the fracture. The flaps were not closed immediately owing to the curious myxomatous appearance of the

muscles The patient made a slow but uneventful recovery Large pieces of necrotic muscle from as high as the tuberosity of the tibia separated during the following weeks

The condition was that of contusion and thrombosis of the anterior and posterior tibial arteries and their venæ comites, due to the injury sustained at the time of the accident

POST-WAR CASES

In a personal communication Mr Watson Jones kindly gave me the following details of his case —

Case 3 —A woman of 32 had been treated for diabetes over several years On Jan 5, 1931, she sustained a comminuted spiral fracture involving the lower two-thirds of the tibia and fibula, with a Pott-Dupuytren of the ankle The fracture was reduced and put in plaster to the upper thigh

Jan 8 —Her toes became insensitive, but there was still a fairly good circulation in them

Jan 9 —Her toes became cyanosed and the front half of the plaster was removed

Jan 10 —There was definite onset of dry gangrene of the toes, but no evidence of pressure sore or plaster pressure was found Peri-arterial sympathectomy of the femoral artery in Scarpa's triangle was performed under local anæsthesia

Jan 11 —There was no improvement in the circulation, and a line of demarcation appeared at the junction of the middle and lower two-thirds of the leg, but it was insensitive as high as the junction of the middle and upper thirds

Jan 12 —The leg was insensitive to 2 in below the knee, and amputation through the lower third of the thigh was performed under gas and oxygen

The patient made a good recovery and now walks well with an artificial limb When the treatment was commenced there was glycosuria and a high blood-sugar, but these were controlled with insulin and glucose The gangrene was dry and the first sign occurred four days after the fracture There were arteriosclerotic changes in the vessels, even the femoral artery in Scarpa's triangle was hardened

Mr Watson Jones further writes "No doubt in this case the diabetic factor was as important as the traumatic factor Apart from this, I know of no case (i.e., of gangrene following a fracture), either personally or in the literature On the other hand, I suppose that fractures of the neck of the femur occurring so frequently in old people are likely now and again to coincide with a case of senile gangrene of the foot Although it would seem probable that the combination of two senile conditions should not be uncommon, I have not actually met a case"

OTHER POST-WAR REPORTS

The literature on fractures complicated by gangrene since the War is limited, though in the latter half of the nineteenth century a good deal was written on the subject, chiefly in Germany

Case 4 (Gregora,² 1927) —A 27-year-old tradesman sustained a supramalleolar fracture of the tibia and fibula in a motor and train accident on Oct 21, 1923 He had a painful hæmatoma in the popliteal space, more towards the outer side Two days later the left foot was cold to the fork of the malleoli, anæsthetic, and showed a few red-blue spots Gangrene of the foot set in during the next few days The patient refused the proposed amputation Digital evacuation of the hæmatoma in the popliteal space was performed and a pyrexia followed On Nov 5 the leg was amputated four finger-breadths above the ankle-joint

The flesh in the amputated foot was suffused with blood Neither the posterior tibial nor the peroneal artery was injured at the site of the fracture The musculature in the remaining part was jelly-like and light grey in colour The rise in temperature continued and much necrotic substance separated from the stump On Dec 11 re-amputation four finger-breadths higher was carried out At the second operation a few yellow gelatinous particles were still present in the muscle Recovery followed

(NB —The gangrene here does not appear to be directly related to the fracture itself, but to contusion of the popliteal artery or of the origin of the anterior tibial artery, sustained at the same time as the fracture)

Case 5 (Gregora,² 1927)—A 17-year-old shop-assistant, on July 4, 1925, was thrown out of a swing-boat when it was at the highest point of its swing, she fell head first to the ground. The right thigh was severely swollen by a hæmatoma at the middle and lower thirds, and a supracondylar fracture of the femur was sustained. From the swelling downwards was an almost complete loss of sensation. The pulse in the dorsalis pedis was not discernible. The blood-clot was immediately cleared out and the artery exposed in the adductor canal. It was found obliquely torn through, and the vein also showed a lentil-sized hole. A circular suture was inserted into the artery after freshening the ends, and a silk suture into the vein. At the site of the suture in the artery, thrombosis occurred immediately, so that the artery was only clear for a short time. As the bone fragments were difficult to retain, they were fixed with a wire band through another incision.

About the fourth day gangrene of the foot set in from the ankle downwards. Only on July 21 was consent for the amputation given. It was performed four finger-breadths above the ankle, and recovery followed.

Case 6 (Gregora,² 1927)—On May 16, 1915, a 56-year-old servant caught her right arm in a washing machine. She received a complicated fracture of the upper and lower arm. Immediately after the injury there was loss of sensation in the fingers, and disappearance of the radial pulse. In the days following, the hand became gangrenous and the lower arm to the elbow-joint was coloured blue. On May 24 amputation was performed in the region of the elbow.

Case 7 (Auvray,⁴ 1924)—On Nov. 13, 1923, a 54-year-old woman was admitted with a fracture of the surgical neck of her humerus. The right radial pulse was diminished at first but returned the next day. There was considerable painful œdema and extensive bruising around the fracture. On the fourth day severe pain in the entire arm occurred and brown patches appeared on the back of the fingers. She had a temperature of 38°–39° C. The patches spread to the hand, and the radial pulse was not palpable. Gangrene of all the fingers and the back of the hand started on the sixth day after injury. The diagnosis was thrombosis of the axillary artery secondary to contusion by the fracture.

At operation the brachial artery was exposed at the neck of the humerus. The cellular tissue around it was echymosed. Pulsation was present above the fracture, but not on a level with it. The inferior scapular branch was intact, the circumflex arteries were seen, and also the profunda brachialis. The artery was opened at the level of the fracture and a clot evacuated, it had prolongations into the circumflex arteries. The arterial intima was striated and had lost its sheen. An adherent clot was also removed. The artery was then closed with a silk suture.

Steady improvement took place after the thrombectomy, but the gangrenous patches remained stationary and mummified. On Dec. 31 amputation through the wrist was performed.

On March 4, 1924, the arm was normal in semi-flexion. Sensation gradually returned, but the radial pulse was still absent. The blood-pressure was fair, but not quite so good as in the opposite arm. Pathologically, a rupture of the intima led to thrombosis.

(N.B.—Earlier arterial exploration might have yielded a better result, the severe pain suggested impending gangrene.)

Case 8 (Muir,⁵ 1924)—A man of 36 fractured his fibula at the upper third, associated with an overlying punctured wound which bled copiously. The position was good. Four days later the foot became cold, bluish, and anæsthetic, and in twelve days there was a line of demarcation immediately above the malleoli, and a dry gangrenous foot. A peri-arterial sympathectomy of the femoral artery, in Hunter's canal, twenty-five days afterwards, had no effect on the gangrene.

A month after the accident amputation at the seat of election was performed, and during it no bleeding was noticed from the anterior or posterior tibial arteries, they were filled with organizing thrombus. The patient recovered.

Examination of the leg showed a localized muscular extravasation of blood around the fracture, which had come from a wound in the posterior tibial artery, this vessel was thrombosed from the popliteal downwards, whilst the anterior tibial, which was also thrombosed, appeared to have been bruised and its inner coat damaged. Sections showed the arteries to be healthy. The circulation to the ankle was apparently carried on by the sural branches of the popliteal artery.

Case 9 (Andler,⁶ 1924)—A man of 57 sustained a comminuted fracture-dislocation of the humerus. There was weakness of the arm and impairment of the arterial pulse in the axilla. Operation revealed that the axillary artery was not pulsating in its upper half, and the thrombosed section was resected after ligaturing. Later, the hand and lower third of the forearm became ischæmic, and necrosis appeared. The arm was amputated at the junction of the upper and middle thirds.

Investigation of the amputated limb showed a circular defect in the intima and media, which hung as a flap from its peripheral end, turned in the direction of the blood-stream, and rolled along the lumen (i.e., of the brachial artery).

(N.B.—This suggests, although not definitely stated, that there was an injury to the brachial artery in addition to the resected part of the axillary artery, so that the gangrene would be due to the double lesion.)

Case 10 (Eaves and Campiche,⁷ 1921)—A farmer aged 55, was thrown from a car and fractured the surgical neck of his left humerus. He was seen three days later and considerable displacement was present. At no time could pulsation be felt in the radial, ulnar, or brachial arteries. He could still move all his fingers, but the fifth was white and swollen.

On the twelfth day after the injury a patch of ischæmic necrosis appeared on the ulnar side of the forearm near the wrist. After two unsuccessful attempts at reduction, on the thirteenth day the head of the humerus was excised and the shaft inserted in the glenoid cavity. The ischæmic gangrene increased until the patient lost four fingers with part of the metatarsals, the forearm remaining in a typical state of Volkmann's ischæmic paralysis.

The surgeons report that they "could inspect and feel the thrombosed axillary artery exposed during the operation, it was spindle-shaped, hard, and pulseless, the adventitia was still intact, so that a diagnosis of rupture of the tunica intima and media was made. The thrombus was 2 to 2½ in long, and its removal followed by double ligation of the axillary artery *would have been quite feasible*."

(N.B.—From the authors' description the thrombus would probably be occluding the origins of the circumflex and superior profunda arteries, thus interfering with the essential anastomotic circulation of the arm and accounting for the gangrene. Early exploration would have been valuable, even though the artery thrombosed again, there was the real possibility that the openings of one or of the other of these branches would have remained patent and permitted an adequate anastomotic flow, or have allowed direct circulation until the smaller collaterals opened up.)

Case 11 (Reichle,⁸ 1921)—This is a case of traumatic segmentary vessel spasm. A strong young man sustained a fracture of the middle of the shaft of his femur by having his right leg drawn very sharply backwards and outwards in a railway accident. He was shocked and unable to walk. The upper part of the right leg was swollen and the lower part was cold. There was extensive bruising in the lower third of the thigh. Pulsation in the popliteal and peripheral arteries could not be felt. As it was believed that the right femoral artery was injured, immediate operation was undertaken. The artery was exposed in the popliteal space below, to the middle of the thigh above, but no injury could be established in it, except that it was not pulsating, thus an even higher lesion was suggested. The vessel was therefore exposed in Scarpa's triangle. Once more no abnormality was detected, and careful observation here revealed only very weak pulsation. Owing to the collapsed condition of the patient, the operation was then terminated. The diagnosis lay between traumatic spasm of the femoral artery and a retroperitoneal hæmatoma involving the external iliac artery.

The patient's condition improved after the operation. The right leg became warmer, and the next day its blood-supply was visibly good, and the pulse of the dorsalis pedis was present. Complete recovery followed.

(N.B.—I can find but this one example of traumatic vessel spasm with a fracture, and even this was associated with threatened and not actual gangrene. Nevertheless, I am sure that the wide exposure of the artery and the relief of tension following the trauma was of definite benefit.)

TWENTIETH CENTURY CASES (1900-14)

There are six of these cases, the two published by Ormsby are here described—

Case 12 (Ormsby,¹ 1911)—Mr J P, aged 20, single, fractured his left femur about the middle third. He complained bitterly of the pain in the left leg and foot at the time of the accident (This is of diagnostic significance with regard to impending gangrene.)

The dorsalis pedis and posterior tibial arteries were not examined. Two days later considerable swelling of the leg and a slight discoloration of the toes were discovered, previously they had been white and cold to the touch. The splints were removed and the limb was placed between sand-bags. The swelling made it difficult to examine the arteries. By the fourth day the discoloration had advanced up the leg, and massage was begun twice daily.

On the sixth day the toes were gangrenous and the leg swollen and mottled, but painless. A diagnosis of occlusion of one or both of the arteries at the site of the fracture was made. The patchy gangrene spread irregularly up the leg, and nine days after the accident the limb was amputated through the thigh at the site of the fracture. At the operation the popliteal artery bled, showing that the femoral artery was intact and not injured at the level of the fracture.

The patient had a secondary hæmorrhage a few days later, the flaps were grossly infected and the thigh became mottled as the leg had been before operation. He died twenty-five days after the accident.

Dissection of the anterior tibial artery from its point of emergence at the interosseous membrane to the ankle showed it to be normal, and fluid injected here appeared from all its smaller branches. Ormsby states, "this demonstration taken in connection with the fact that at the time of amputation the popliteal bled despite the tourniquet, led me to conclude that the cause of the gangrene was not arterial."

(N.B.—Ormsby makes no mention of the actual bifurcation of the popliteal artery where the block may have been, and which, if present, would have occluded the posterior tibial artery more than the anterior (the former being the larger and straighter vessel). The thrombus would probably have been astride the bridge of the V behind the interosseous membrane, and consequently it would not have shown in the dissection of the anterior tibial in the front of the leg. Again, the bleeding popliteal artery, especially noted by Ormsby was, by means of its anastomotic vessels around the knee, quite compatible with an obstruction at its termination.)

He further states that some of the veins seemed to be entirely normal and were flaccid, containing a small quantity of fluid blood, whilst others were filled to bursting with clotted blood, this not throughout their entire extent, but only under the gangrenous spots. He decided that the gangrene was due to venous thrombosis, the result of a phlebitis induced by the external violence done to the part at the time of injury. He concluded that in fractures of the femur help in the prognosis is available by examination of the arteries of the leg and foot.)

Case 13 (Ormsby,¹ 1911)—A man aged 45, an alcoholic subject, infected with syphilis ten years previously, sustained a fracture of his left femur at the middle third, in a train accident. It was manipulated and splinted in flexion with extension. Two weeks later moist gangrene of the leg spread well up towards the knee, but it was not continuous, there were scattered patches of necrotic tissue without apparent definite distribution. It extended deeply into the calf of the leg, the gastrocnemius and soleus sloughed away and exposed the tibia for 3 to 4 in. Amputation above the knee was carried out. The femoral artery and vein appeared to be normal. Unfortunately, the amputated limb was not dissected, and thus the cause of the gangrene was not determined.

The patient recovered. The possibility of a syphilitic disease of the artery must be considered here.

NINETEENTH CENTURY CASES (1850-1900)

There are twenty cases of gangrene following a fracture recorded in this period. I have tabulated their essential details, and I include brief descriptions of the more interesting reports. It should be remembered that many of them would be treated by the pre-Listerian technique, and consequently the results are not strictly comparable with the more recent ones already reviewed.

Case 14 (Chuquet,⁹ 1877) —A man aged 65 was run over by a cab in Paris on Feb 26, 1877. He had a compound comminuted fracture of the left tibia and femur, with openings into the knee-joint at its inner and outer borders. There was not much bleeding.

On the patient's admission to hospital his leg was white and cold, it remained so in spite of efforts to warm it, no pulse being felt in the dorsalis pedis or posterior tibial arteries. Sensation, however, was not altogether lost, the patient could feel pin-pricks distinctly. His general condition was otherwise satisfactory, although he was an habitual drunkard, and a few days previously he had been involved in another accident, when he fractured a rib.

The next day the wound smelt offensively and the tissues gave the sound of emphysema and crepitus on percussion. (This was almost certainly a gas gangrene infection, although it is not mentioned as such in the article.) The foot was now warmer, but not so warm as the right. Neither pulsation nor sensation was detected in the leg. Amputation through the thigh was performed. The bleeding was free and not controlled by compression, since the arteries were so atheromatous.

Two days after the injury there was a foul-smelling discharge from the wound, and a day later the patient died.

Post-mortem examination of the popliteal artery, vein, and their branches showed them to be intact. The artery was atheromatous. In its upper portion was a white fibrous clot, 1 cm long, firmly adherent to the wall, but it was not completely obstructing the lumen. (This was probably caused by the previous accident, for, as the limb was amputated the day after the fracture, the period was too short for fixation and organization of a thrombus caused by the second mishap.)

There were two more clots at the bifurcation of the popliteal, an upper one about 4 cm in length and non-adherent, whilst below it and without doubt detached from the former, of the same age and form, was another clot spreading into the anterior and posterior tibial arteries. Careful examination of the arterial wall at this level showed that the intima had been ruptured for 7 to 8 mm, almost encircling the artery.

(N.B.—Whilst the patient probably died from the toxæmia of a gas-forming infection, the gangrene of the limb was due to thrombosis and embolism of the popliteal artery at its bifurcation, the thrombus having arisen at the site of a contusion in an already atheromatous artery. Chuquet comments that a similar case had not been previously recorded, and that it was a rare occurrence—i.e., fifty-seven years ago.)

Case 15 (Jungst,¹⁰ 1882) —H.M., a man aged 20, sustained a compound fracture of the internal condyle of the tibia and head of the fibula on March 17, 1882. The posterior tibial and dorsalis pedis pulses could not be felt. The left foot was cold although sensibility was normal. On the third day it was anæsthetic and subsequently gangrenous.

At the investigation it was found that the media of the popliteal artery was torn for 2.5 cm at a point 2.5 cm above the bifurcation, the intima was also torn for 5 mm, whilst the artery itself was narrowed and thrombosed. Diagnosis: contusion and thrombosis of the popliteal artery.

(N.B.—The narrowed artery in this patient corresponds with Stopford's¹¹ finding in a case of senile gangrene of the foot reported in the *BRITISH JOURNAL OF SURGERY*. The two illustrate a predisposing factor in the incidence of gangrene of the leg after middle life, i.e., a narrowed and thickened popliteal artery.)

Case 14 (Jungst,¹⁰ 1883) —A man aged 19 sustained a simple fracture of his forearm in a machine accident on Nov 11, 1883. At first there were only slight symptoms with swelling, but by the fourth day the forearm was ischæmic and the radial and ulnar pulses had disappeared, nor did they return. Gangrene occurred on the sixth day and the arm was amputated.

On examination the brachial artery was apparently intact but thrombosed, its intima was torn for half its circumference 3 cm above the bifurcation, and the thrombus was adherent to this. The brachialis anticus and joint capsule were also torn at this level. The elbow-joint was full of blood. Diagnosis: contusion and thrombosis of the brachial artery just above the elbow-joint.

(N.B.—The swelling in and around the joint would damage the anastomotic vessels, so that, combined with the cessation of the main blood-supply, gangrene was inevitable.)

Table I—ANALYSIS OF CASE

	AUTHOR OF CASE	AGE	SEX	BONE FRACTURED	CONDITION ARTERIE
1850-1900	Hawkins ¹⁴ (1850)	28	M	Humerus middle third Open	Not noted
	Holt ¹⁴ (1851)	47	M	Humerus upper Comminuted	Not noted
	Cadge (1859)	30	M	Femur	Not noted
	Broca ¹⁴ (1862)	43	M	Femur Compound comminuted	Not noted
	Engelman (1862)	20	M	Femur lower third Simple	Not noted
	Billroth (1863)	23	M	Femur lower third	Not noted
	Mash ¹⁴ (1872)	38	M	Dislocated and fractured humerus Supra-coracoid	Not noted
	Defrance ¹⁴ (1873)	51	M	Femur Supracondylar	Atheromat
	Lucke (1873)	Not noted	Not noted	Femur Double	Not noted
	Hulke ¹⁴ (1875)	58	M	Radius with torn muscles Compound	Veins throbosed
	Bimbenet ¹⁴ (1877)	Not noted	M	Humerus with crushing	Not noted
	Chuquet (1877)	65	M	Femur condyles Tibia tuberosities	Atheromat
			Drunken	Compound	
	Rivington ¹⁴ (1878)	19	M	Femur condyle Tibia head	Not noted
	Jungst (1882)	20	M	Splint head of fibula, end of tibia Slightly compound	Not noted
	Jungst ¹⁴ (1883)	19	M	'Forearm' Simple	Not noted
	Broca ¹⁴ (1885)	27	M	Femur lower part Tibia upper part	Healthy
				Complicated by much crushing	
	Rosenberger ¹⁴ (1887)	50	M	Radius	Not noted
	Merkel ¹⁴ (1887)	Not noted	M	Radius and ulna Dislocated wrist Splintered	Not noted
1900-1914	Schulz (1897)	32	M	Tibia lower and middle third Extensive bruising	Syphilitic
	Stromeyer ¹⁴ (1899)	Not noted	Not noted	Humerus Simple	Not noted
	Muller (1911)	Not noted	Not noted	Femur lower and middle thirds	Not noted
	Selenow (1911)	Not noted	Not noted	Femur	Not noted
	Burr (1911)	Not noted	Not noted	Femur	Not noted
	Weinlechner (1911)	Not noted	Not noted	'Fracture'	Not noted
	Ormsby (1911)	45	M	Femur middle third Simple	Syphilitic + alcoholic
	Ormsby (1911)	20	M	Femur middle third Simple	Not noted
	Reichle (1921)	Not noted, young	M	Femur middle Simple	Healthy
	Eaves and Campiche (1921)	55	M	Humerus surgical neck Much displacement Simple	Not noted
	Auvray (1924)	54	F	Humerus surgical neck Simple	Not noted
	Andler (1924)	57	M	Comminuted with dislocated humerus Simple	Not noted
	Muir (1924)	36	M	Fibula upper third Compound	Not noted
	Gregora (1927)	27	M	Tibia and fibula lower third Simple	—
	Gregora (1927)	17	F	Femur middle and lower third Simple	—
	Gregora (1927)	56	F	Humerus, radius, and ulna Simple	—
	Watson Jones	32	F	Spiral of lower tibia and Pott-Dupuytren of ankle Simple	Diabetic arteritis
	Dodd	59	M	Femur lower third Compound	Calcified
	Dodd	29	M	Tibia and fibula middle Simple	Normal

LOWING FRACTURES

DIAGNOSIS OF VESSEL INJURY	OPERATION	RESULT
Contusion and thrombosis of brachial artery	Amputation 5th day	Not noted
Contusion and thrombosis of axillary artery	Amputation to shoulder	Not noted
Torn popliteal artery	Amputation 8th day	Not noted
Thrombosed popliteal and femoral veins	Amputation below knee	Died tetanus
Popliteal artery obliterated		
Aneurysm of lower femoral artery	Ligature of artery	Died 7th day
Torn and thrombosed femoral artery	Amputation lower third of femur on 16th day	Died pyæmia
	—	Died 4th day
Torn axillary artery at origin of subscapular artery		
Thrombosed popliteal at level of fracture	No amputation	Died 25th day
Torn femoral artery	—	Died
Thrombosed brachial artery, torn in lower third	—	Died 7th day
Contused and thrombosed brachial artery	Arm amputated	Not noted
Thrombus and embolus of bifurcation of popliteal artery	Amputation thigh 8th day	Died gas gangrene
Torn popliteal artery and vein	Amputation 2nd day	Not noted
Contusion and thrombosis of popliteal artery (torn, narrowed, and thrombosed above bifurcation)	Amputation lower thigh 8th day	Not noted
Contusion and thrombosis of brachial artery (torn intima, much peri-articular swelling)	Amputation of arm	Not noted
Torn, divided, and thrombosed posterior tibial artery	Amputation of thigh after 5 hours	Died 35 hours
Torn intima with thrombosis of brachial, radial, and ulna arteries	Amputation above condyles of humerus	Not noted
Vessels not investigated	Arm amputated	Not noted
Torn and thrombosed popliteal artery and vein	Amputation at femur after 1 day	Recovered
Contusion and thrombosis of brachial artery	Amputation through arm on 2nd day	Not noted
Rupture of middle and inner coats, and thrombosis of the lower part of femoral artery	Amputation thigh	Died
Laceration of femoral vein	Amputation thigh	Died 'officious treatment'
Contusion and thrombosis of femoral artery	Amputation thigh	Not noted
Rupture of artery and vein	Amputation	Died
Diffuse arterial hæmatoma of popliteal artery	Amputation through thigh	Recovered
Thrombosis + embolism (2) of the bifurcation of the popliteal artery	Amputation through thigh	Died
Traumatic segmentary spasm of femoral artery	No amputation	Recovered
Thrombosed third part of axillary artery for 2 to 2½ in	Lost 4 fingers, part of metacarpals Ischæmic contracture of forearm	Recovered
Contusion and thrombosis of axillary artery	Embolectomy on axillary artery Amputation through wrist	Recovered
Contusion and thrombosis of brachial artery	Amputation	Recovered
Contusion and thrombosis of posterior and anterior tibial arteries	Amputation at 'seat of election'	Recovered
Thrombosed popliteal artery	Amputation middle of leg and higher later	Recovered
Contusion and thrombosis of femoral artery	Amputation through lower quarter of leg	Recovered
—	Amputation through elbow	Not noted
Probably thrombosis of anterior and posterior tibial arteries	Peri-arterial sympathectomy Scarpa's triangle 6th day Amputation thigh 8th day	Recovered
Embolus at bifurcation of popliteal artery	Amputation through lower thigh	Recovered
Contusion and thrombosis of anterior and posterior tibial arteries	Amputation of leg at seat of election	Recovered

THE CASES ANALYSED

Table I is a general analysis of the cases of gangrene following fractures

Age and Sex Incidence (*Table II*)—Of 31 patients, 27 were men and 4 were women, thus men easily predominate. The largest group (11) occurs in males between the ages of 15 and 30 years, this is the most active age-period. At the other end of the scale there are nine patients over 50, of whom two are women.

Table II—ANALYSIS OF THE AGE AND SEX GROUPS

AGE GROUPS	15-30 YEARS	31-45 YEARS	46-60 YEARS	61-75 YEARS	NOT NOTED	TOTAL
Men	11	5	7	1	3	27
Women	1	1	2	—	—	4
Total	12	6	9	1	3	31

The Bones Affected—The lower limb was affected 23 times, distributed as follows —

The femur 16 (lower third)
 The tibia 2
 The fibula 1 (upper end)
 The tibia and fibula 2
 The femur and tibia 2

The upper limb was involved 12 times, divided thus —

Humerus 7 (upper half)
 Radius and ulna 4
 Radius, ulna, and humerus 1

Table III—ANALYSIS OF THE LESIONS AND THEIR LOCATION

DIAGNOSIS	TOTAL	FEMORAL ARTERY	POPLITEAL ARTERY	ANTERIOR AND POSTERIOR TIBIAL ARTERIES	THIRD PART OF THE AXILLARY ARTERY	BRACHIAL ARTERY	REMARKS
Embolism of main artery	3	—	3	—	—	—	—
Contusion and thrombosis of main artery	20	4	3	3	4	6	—
Complete tearing and thrombosis of main artery	10	3	4	1	1	1	—
Diffuse traumatic aneurysm	1	1	—	—	—	—	—
Traumatic segmentary vessel spasm	1	1	—	—	—	—	—
Injuries, nature doubtful	2	—	—	—	—	—	(1) Lacerated femoral vein (2) 'popliteal' artery
TOTAL		9	10	4	5	7	

Thus gangrene is most likely to follow fractures of the proximal half of the humerus in the arm, and those of the distal third of the femur in the lower limb

Predisposing Factors—In 5 cases these were mentioned They are calcification of the artery in 2, syphilis in 2, and diabetes in 1 As 9 of the patients were over 50, there would probably be some degree of arterial disease present in them

The Exciting Causes of the Gangrene—*Table III* shows the exciting causes of the gangrene and indicates that the femoral, popliteal, and brachial arteries are most liable to injury in association with fractures

The Type of Gangrene—This was dry in 6 cases, wet in 3, and 'threatening' in 1 In the remainder of the series its nature was not stated

The Time of Onset of the Gangrene after the Injury—Throughout the series it occurred during the third to the sixth day after the actual injury, usually on the fourth

The Results—Amputation was performed on all but four occasions The site of this was through —

The thigh in 14
The leg in 5
The arm in 8

The wrist in 1
The hand in 1
The place was not specified in 2

Table IV shows an analysis of the results

Table IV—ANALYSIS OF THE RESULTS

	1918-32	1900-14	19TH CENTURY	TOTAL
Total cases	10	7	20	37
Cases recovered	9	1	1	11
Cases died	—	4	9	13
Results unknown	1	2	10	13

Thus, stated baldly, out of 24 cases, 11 recovered and 13 died, so that there is apparently a heavy mortality of 54 per cent

However, further investigation reveals the interesting significant facts that since the War, of 9 cases fully reported, all have recovered Between 1900 and 1918, 4 out of 5 cases died—an 80 per cent death-rate, except that in two patients the result is not stated, but nevertheless 4 deaths in 7 cases is very high Before 1900, 9 out of 10 cases succumbed, of these last fatalities, probably most were due to infection of the wound received at the time of the injury or at the amputation Tetanus is mentioned once or twice, but antitetanic serum was not then known Lister's technique was only in its infancy during some of this period and was not in general use Since the opening of the antiseptic or aseptic era the mortality has strikingly fallen to the negligible figure of the present day—an eloquent testimony to the life-saving value of Lord Lister's work

DISCUSSION

1 **The Interruption of the Blood-supply**—Gangrene may follow after several types of injury to the main artery of the limb These are —

a Complete division of the chief vessel by sharp or blunt trauma

b Penetration of the principal artery, e.g., by a fragment of bone

Groups *a* and *b* usually result in cessation of the blood-supply by thrombosis or by the formation of a diffuse traumatic aneurysm

c Contusion or rupture of the intima, or of the media and the intima together, leading to vascular obstruction by thrombosis

d Embolism by a thrombus which has originated in the artery at the level of the injury becoming detached and lodging more distally as an embolus, it is usually located at a bifurcation, e.g., at that of the popliteal artery

Prognostically, Group *a* is more favourable than Group *c* (Gregora²), as in the latter there is more likely to be coincident injury to the tissues, including the anastomotic vessels

e A condition of segmentary vessel spasm may also result in threatened or actual gangrene, it was apparently first described by Finalij¹² The spasm lasts up to twenty-four hours In one of Finalij's patients peripheral gangrene occurred I have only been able to find a single true example of this affection associated with a fracture (*Case 11*), gangrene did not actually happen, but definitely threatened The prompt operation probably played a part in averting it (Reichle⁸)

In addition to the above accidents to the main vascular supply, all degrees of trauma to the tissues, to the nerves, to the lymphatics, and to the smaller vessels are associated In a good subject the lesser arteries if intact will easily carry on the collateral circulation, but if, in addition to the main vessel lesion they too are involved, then gangrene is almost inevitable

Factors of the general health also predispose to and influence the amount of and the incidence of the gangrene which occurs, e.g., diabetes, syphilis, or alcoholism, and, more particularly, the local state of the arteries, such as narrowing or calcification (*see Figs 163, 164*)

2. Why Certain Vessels are Injured—An explanation for the apparent frequency of damage to certain blood-vessels by fractures is worthy of note Briefly, the underlying principle is that the more mobile the vessel, the less likely it is to be involved by accidents On the other hand, the more fixed an artery is by fascia, by aponeurosis, by proximity to bone, or by several branches arising close together, the more likely it is to be hurt, for it is unable to slip away from the violence (*Figs 168, 169*)

THE CHARACTERISTICS OF THE INJURED VESSELS —

a The Lower Third of the Femoral Artery—This is restrained as it passes close to the femur under the tough fibrous arch in the adductor magnus Above this, the artery is embedded in Hunter's canal and is more removed from the bone

b The Bifurcation of the Popliteal Artery—This is anchored by the fibrous arch of the soleus, by the passage of the anterior tibial artery over the interosseous membrane, by its proximity to the fibula, and by the origins of small anastomotic branches to the knee-joint (*Fig 168*)

For the cross-section through the termination of the popliteal artery (*Fig 168*) I am indebted to Professor Harris, of University College Hospital He very kindly sectioned a leg which was photographed, such a diagram is, I think, unobtainable from the text-books He pointed out the comparatively intimate association of the bifurcation with the neck of the fibula He showed me radiograms demonstrating

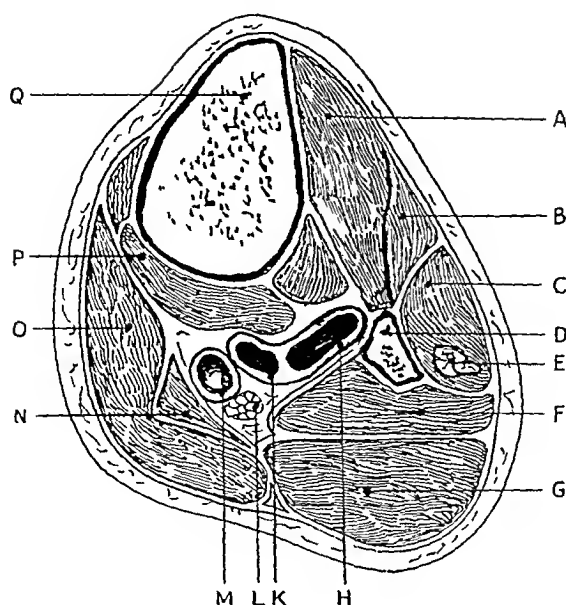


FIG 168 —Photographic section through the upper third of the right leg at the level of the bifurcation of the popliteal artery, this is at the origin of the anterior tibial artery, as it arises from the termination of the popliteal artery and runs horizontally forward to the anterior compartment of the leg in close proximity to the fibula A, Tibialis anticus, B, Extensor digitorum longus, C, Peroneus longus, D, Fibula, E, External popliteal nerve, F, Soleus (fibular head), G, Gastrocnemius, H, Anterior tibial artery, K, Termination of the popliteal artery and beginning of the posterior tibial artery, L, Internal popliteal vein, M, Internal popliteal nerve, N, Soleus (tibial head), O, Gastrocnemius, P, Flexor longus digitorum with tibialis posticus antero laterally, Q, Tibia

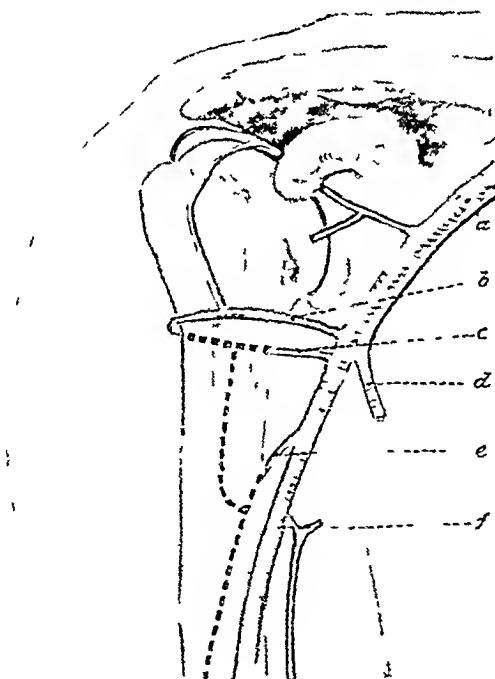


FIG 169 —Semidiagrammatic drawing of the third part of the axillary artery and the beginning of the brachial artery, illustrating the origins of the subscapular artery (*d*), the anterior and posterior circumflex arteries (*b* and *c*), and the profunda brachii (*e*). It shows the consequent fixation of the axillary artery, and quick diminution in the calibre of the beginning of the brachial artery *a*, Axillary artery, *b*, Anterior circumflex artery, *c*, Posterior circumflex artery, *d*, Subscapular artery, *e*, Profunda brachii artery, *f*, Brachial artery

on the inside of the neck of the fibula a slight groove which is made by the anterior tibial artery as it passes from behind to the front compartment of the leg. This emphasizes the vulnerability of the artery to injury in fractures of this region of the fibula.

c The Anterior and Posterior Tibial Arteries—These are also close to the fibula, more especially in the upper half of the leg, and further they lie on or are covered by aponeurosis. Professor Harris pointed out that the anterior and posterior tibial arteries are closer to the fibula than to the tibia. He suggested that "anterior and posterior fibular arteries" would be a more correct name for them. Muir's case (*Case 8*) of a fracture of the upper third of the fibula followed by gangrene illustrates this point.

d The Third Part of the Axillary Artery and the Brachial Artery (Proximal Portion)—The axillary artery is comparatively fixed and somewhat tied by the origins of its branches, the subscapular, the anterior and the posterior circumflex arteries, arising close together (*see Fig 169*) (Gregora²). Below this the brachial artery is mobile, so that, as is to be expected, the injury occurs at the junction of the mobile and relatively immobile parts.

THE DIAGNOSIS OF INJURY TO AND OCCLUSION OF A MAIN ARTERY

All or various combinations of the following signs and symptoms may be found—

1 *Arterial Pulsation*—The presence of pulsation above the injury, with impairment of or loss of it below this level. This characteristic may be of immediate or gradual onset, it is a dependable and important one. Of course comparison with the normal (i.e., with that of the uninjured limb), is necessary, for the poor general condition of the patient may explain the weak pulse.

2 *Sensation*—Below the traumatized place an alteration from the normal sensation is present. It may be anæsthesia or paræsthesia, or, on the contrary, severe persistent pain localized about the seat of the damage, or even distributed throughout the entire limb. When the latter occurs, especially after reduction and splinting, I consider gangrene is very likely (Auvray's and Ormsby's cases).

3 *Appearance*—Blanching, cyanosis, and 'stone' coldness of the limb below the hurt area. Later bullæ and large blebs may form on the skin.

4 *Paralysis*—Loss of muscle power, e.g., inability to make the slightest movement of the foot or toes after a fractured tibia, even with considerable effort. Muscle sense also seems to be impaired.

5 *Hæmatoma*—The presence of a hæmatoma and bruising about the vessel. This may not be immediately apparent on inspection, but if the part is palpated by the fingers, the tension will be detected. When the hæmatoma is under the deep fascia, then staining from it will not show until a few critical days have passed, and during this time gangrene may become established.

6 *Local Tenderness*—Tenderness and pain localized precisely over the chief vessel at the point of injury is sometimes found—it suggests thrombosis. However, in a deeply situated artery like the popliteal, in a stout patient, or when there is considerable traumatic swelling, this feature will not be found. If similar localized tenderness and thickening is observed over the artery where it narrows or bifurcates distal to the injury, an embolus there is suggested.

7 *Blood-pressure*—A gradual fall in the blood-pressure of the limb as compared with the systemic figure denotes a failing local circulation (Auvray⁴)

8 *Radiograms*—A careful inspection of the radiograms may reveal signs of calcified arteries. In this event little vascular compensation is to be expected after arterial injury, and extensive gangrene is probable. This peculiarity indicates a high level for the amputation in order to ensure well-vascularized flaps and consequent sound healing (See Fig 163)

THE TREATMENT OF THREATENED GANGRENE

When the onset of gangrene following a fracture is suspected, in view of the inevitable amputation when it occurs, surgical interference even though it may be heroic is justified. "Look and see" rather than "wait and see" is then quite rightly the surgeon's governing principle. That all fractures should be reduced and suitably splinted as soon as possible after their occurrence is a *sine qua non*. The standing order in force in some hospitals, that this treatment must be carried out within four hours of the patient's admission, is worthy of general adoption.

The systemic indications of diseased arteries, and especially the previously described signs of interference with the circulation in a limb, will be watched for in all bone injuries. Assistants will be instructed to look for and to report them. The urine is examined for sugar.

When the suspicion of impending gangrene has arisen, a definite intimation of progress to recovery or to gangrene is sometimes obtainable by recording on the skin in coloured inks, say at four-hourly intervals, the levels of (1) Altered sensation (or anæsthesia), (2) Modified temperature, (3) Variation in colour. Temperature is indicated by black ink, sensation in red, and colour by a blue skin-pencil. Each line is appropriately labelled with the time of the observation (Mr Watson Jones's case illustrates the value of this)

When the symptoms have appeared and have not shortly subsided, wide exploration of the main vessels at the site of injury is promptly suggested. The objection that by such a procedure a simple fracture is converted into a compound one, with its attendant risks of sepsis, is with modern surgical technique fortunately a small and negligible one when compared with the possible loss of a limb by gangrene. In a compound fracture free 'opening up' of the traumatized area by promoting drainage probably lessens rather than increases the sepsis. According to Bohler¹³ it is the best preventive of tetanus and gas gangrene.

During the investigation of the vessels, whether they are found normal or damaged, hæmatomata will be evacuated, and the additional valuable effect of reducing tension in the traumatized tissues will be achieved. The latter will relieve lymphatic and venous stasis and permit the resumption of these circulations, whilst the formation of the collateral anastomosis by the smaller arteries will be assisted. The pressure of exudate and of extravasated blood may be sufficient to prevent totally or partially the development of these lesser compensatory circulations.

When the vessels have been defined the further procedure will depend on the findings, on the patient's condition, on the surgeon, and on his equipment. One of several operations may be necessary—

1 Torn or divided arteries will call for repair, either by ligature of the ends, or, if possible, by excision of the injured section followed by end-to-end

anastomosis The latter is only possible when a short length of the vessel is injured It aims to achieve the ideal, and occasionally it will be successful

2 The artery may be found apparently uninjured but thrombosed—one or two cases in this series were so affected The vessel is temporarily occluded above and below the block (a tape ligature does this satisfactorily), and it is then opened longitudinally and the thrombus is removed The upper ligature is now released in order to establish that all the clot has been removed and that the circulation is present from above Occasionally more clot is washed out by the blood-stream by this manœuvre The artery is controlled again and the lower tape is similarly dealt with If blood flows from below (this will be a rare occurrence after an accident), it will show that the collateral circulation is working, and the continuity of the arterial lumen will be established The wound in the vessel is sutured with the finest vaselined silk, threaded in a slender eyeless needle, as used for ophthalmic surgery

When the artery is opened, the intima or media may be seen to be damaged, e g, a large tear, or even a flap of it may be detached, if the occasion is suitable, the injured section is excised and an end-to-end anastomosis performed

No attempt is made to close the wound, it is made of sufficient extent to gape open, and is dressed with gauze and sterile vaseline, magnesium sulphate, and glycerin paste, or 1-1000 acriflavine and paraffin emulsion (Boots) Silkworm gut sutures may be inserted and knotted at their extremities, when, should the limb live and the progress of the wound be satisfactory, they are tied four to seven days later If immediate closure of the wound is considered advisable, then around it several incisions extending through the deep fascia should be made for drainage and to relieve and prevent tissue tension by extravasations and traumatic œdema

3 If the artery is normal at the level of the injury, the possibility arises of a thrombus having formed and passed distally as an embolus When this rare event is present, embolectomy at the affected point is indicated *Case 1* illustrates an opportunity for it

Careful inspection over the surface markings of the artery will occasionally reveal signs indicating an impacted embolus The common sites for them to lodge are at the termination of the axillary, and at the bifurcations of the brachial and of the popliteal arteries The exposure of the termination of the popliteal artery is a large dissection, but it is certainly worth while, and the necessary extensive muscle-splitting should be confidently undertaken (*see Fig 168*) Palpation by the tip of the finger of such a vessel will reveal the obstruction as a firm thickening, with pulsation above the point and a silent contracted artery below it

4 When a diffuse traumatic aneurysm has formed, a tourniquet is applied above the swelling If the lesion is near to the trunk this is not always practicable, so the main artery is exposed and temporarily controlled by a tape ligature through normal tissue closely above the lesion The aneurysm is incised approximately in the line of the surface marking of the main vessel and the blood-clot evacuated The space is irrigated with hot or iced saline, and an attempt is made to define and suture the artery as the circumstances indicate

Further, the work of the late Sir George Makins³ showing the undoubted benefit of ligature of the main veins of a limb when the chief artery is injured,

should, I think, be followed in each case, but frequently the vein is already thrombosed, so that the need does not always arise

Peri-arterial Sympathectomy.—If the patient's condition permits, some form of peri-arterial sympathectomy is performed on the healthy artery above the level of the injury at the same time as the previous explorations. It will probably have a favourable effect by the resulting temporary vasodilatation existing long enough to nourish the limb through a few critical hours

Finally—and this point is worthy of stress and of repetition—surgery, to be effective, should not be delayed, it must be thorough and extensive, and undertaken in a spirit of optimism. Four to six hours would be the longest period to watch a suspect limb before interference, for a careful exploration is very unlikely to have any other than a good effect

SUMMARY

- 1 The recorded cases of non-infective gangrene following various fractures are described and tabulated
- 2 The causes and symptoms of threatened gangrene are discussed
- 3 A method of treatment in the event of the onset of gangrene being suspected is detailed

REFERENCES

- ¹ ORMSBY, *Internat Jour Surg*, 1911, Aug, 238
- ² GREGORA, *Beitr z klin Chir*, 1927, cxi, 199
- ³ MAKINS, *On Gunshot Injuries to the Blood-vessels founded on Experience gained in France during the Great War*, 1919 Bristol John Wright & Sons Ltd
- ⁴ AUVRAY, *Bull et Mem Soc nat de Chir*, 1924, l, 346
- ⁵ MUIR, *Lancet*, 1924, ii, 321
- ⁶ ANDLER, *Arch f klin Chir*, 1924, cxxxi, 140
- ⁷ EAVES and CAMPICHE, *Ann of Surg*, 1921, lxxiv, 620
- ⁸ REICHLE, *Beitr z klin Chir*, 1921, cxix, 650
- ⁹ CHUQUET, *Progres med*, 1877, v, 350
- ¹⁰ JUNGST, *Beitr z klin Chir*, 1899, xxi, 643
- ¹¹ STOPFORD, *Brit Jour Surg*, 1924, xii, 92
- ¹² FINALIJ, Quoted by Gregora
- ¹³ BOHLER, *Zentralb f Chir*, 1933, May, 1227
- ¹⁴ HERZOG, *Beitr z klin Chir*, 1899, xxi, 643
- ¹⁵ SCHULZ, *Deut Zeits f Chir*, 1897, xlv, 476

CHRONIC EPIDIDYMO-ORCHITIS OR FIBROSIS OF THE TESTICLE OF FILARIAL ORIGIN*

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DURING the last two years I have had the opportunity of examining and treating several cases of chronic epididymo-orchitis of non-venereal origin. I was convinced, after careful urological and bacteriological examinations, that the recurrent attacks of testicular inflammation in these patients were not due to the gonococcus or the *Bacillus coli*. I was then induced to investigate the nature of filarial infection of the genital tracts in the male. It is well known that very often the so-called idiopathic vaginal hydrocele in India, and occasionally sterility, are due to complications of filariasis. During operations on the scrotal tumour marked enlargement of the testicles is frequently met with, and the condition may persist for years after the operation. Little was known about the ultimate fate of such a testicle. Pain of a dull aching character is usually complained of, and on examination the testicle is found to be firm in consistency and heavy. In several cases one testicle was found to be as large as a duck's egg, and occasionally the condition is bilateral. It is obvious that the investigation and elucidation of the pathological relationship of the filaria in chronic enlargement of the testicle are beset with many difficulties. The following case is therefore of great interest.

A J, aged 25, was admitted under my care on April 2, 1933, and discharged on April 30.

HISTORY—The patient complained of persistent pain of one week's duration in the right testicle, which had been getting enlarged for the last five years, but it was not causing much inconvenience to him. There had been no fever. In November, 1932, he had an attack of pain in the testicle with much swelling, which persisted after subsidence of pain in the course of a fortnight. He had another similar attack in February, 1933, resulting in further increase in the size of the testicle.

The patient had suffered both from syphilis and gonorrhœa nearly three years previously. There was no history of aspiration of the affected side for a hydrocele.

ON EXAMINATION—The skin of the scrotum was slightly thickened and there was some scabies, but it was not elephantoid. There was a small hydrocele on the left side. Otherwise nothing abnormal could be detected in the testicle or the cord. On the right side the testicle was much enlarged. It was pyriform in shape, regular in outline, and slightly tender on pressure. On measurement its maximum longitudinal and transverse diameters were found to be nearly 6 in. and 3½ in.

* Being a paper read at a clinical meeting of the Calcutta Branch of the British Medical Association on Aug. 11, 1933.

respectively. It was uniformly firm in consistency except anteriorly at the junction of the lower and middle thirds of the tumour, where it was soft and almost fluctuant and the skin of the scrotum was adherent to the tumour for an area 1 in across. It imparted a sense of weight to the examining hand. Testicular sensation was present. The epididymis could not be palpated separately and there was no vaginal hydrocele. The spermatic cord was nearly twice the normal size in thickness and was lymphangiectatic, but the vas deferens appeared to be unchanged. There was no urethral discharge, and on rectal examination nothing abnormal was noticed. The abdominal lymphatic glands—viz, pre-aortic and iliac—were not palpable. Inguinal glands were palpable on both sides.

The general health appeared to be good, but pyorrhœa alveolaris was present.

On ADMISSION—Temperature 99°, pulse-rate 112

Urine —		Examination of Blood —	
Specific gravity	1010	Leucocytes	10,800
Sugar	Nil	Polymorphonuclears	78 per cent
Albumin	Nil	Lymphocytes	18 „ „
		Large mononuclears	2 „ „
		Eosinophils	2 „ „
Blood-pressure —			
Systolic	110 mm Hg	Malarial parasite	Nil
Diastolic	75 „ „	Microfilaria	Nil
Wassermann reaction	Moderately positive	Prostatic smear	Negative

TREATMENT —

General—Mist pot iodide was given for a fortnight, and the daily dose of the iodide was gradually increased to 45 gr. With regard to scabies, the routine treatment with sulphur ointment was carried out for three days. The patient was referred to the dental department for the treatment of pyorrhœa alveolaris. Glucose was given freely by the mouth before the operation.

Local—The semi-fluctuant area was incised under local anæsthesia and about a drachm of thick turbid fluid was evacuated. It was sterile on culture. The tumour, with all the adherent skin, was removed under local anæsthesia with novocain. The spermatic cord was divided just below the inguinal canal. Lymphangiectasis of the cord was well marked.

Result—The patient had an uneventful recovery and the wound healed by primary intention. On his discharge from the hospital, he was advised to continue the antisyphilitic treatment and to report himself once every month for further examination and for the aspiration of the hydrocele on the other side.

PATHOLOGICAL EXAMINATION —

1 *Macroscopic Appearances*—The large tumour was pyriform in shape and smooth in outline. Anteriorly the skin of the scrotum was adherent at the junction of the middle and lower thirds of the tumour, where there was a necrotic ulcerating

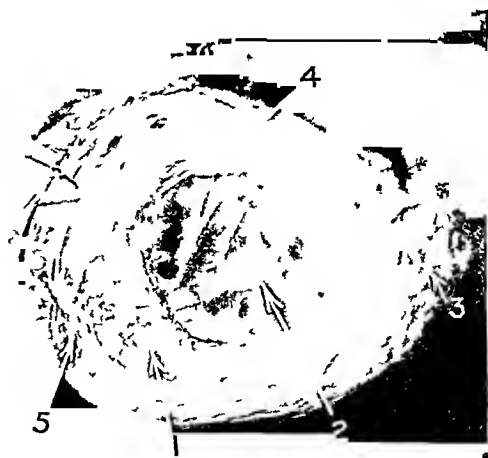


FIG 170—Showing 1, Testis, 2, Epididymis, 3, Spermatic cord, 4, General fibrosis and obliterated vaginal sac, 5, Site of adult filariæ

area with a diameter of nearly $\frac{1}{2}$ in. The skin was thickened in this region, but there was no blubbery tissue characteristic of elephantiasis. The spermatic cord was attached to its upper end posteriorly.



FIG 171—Showing 1, Oblique and transverse section of adult female filaria containing numerous microfilariae within the uterus, 2, Eosinophil granulation tissue surrounding the dilated lymphatic ($\times 270$)



FIG 172—Showing transverse section of the adult filaria, with marked fibrosis and round-cell infiltration ($\times 2$ Zeiss)

On transverse section through the adherent skin, the surface presented a whitish fibrous zone round the periphery, being $\frac{1}{2}$ in thick in some places. The vaginal sac was completely obliterated. The body of the testis and what appeared to be the epididymis were embedded in a connective-tissue matrix. The former

appeared as a lenticular area of greyish colour having been compressed from side to side, but the latter presented a rounded homogeneous gelatinous area of a darker colour, having a diameter of $1\frac{1}{4}$ in. In the cross-section the epididymis appeared to be the larger of the two (*Fig 170*)

2 Microscopic Appearances —

a Testis — The sections through the body of the testis showed scattered areas of round-cell infiltration with a varying number of plasma cells and fibroblasts in the interstitial tissues and in the neighbourhood of the seminiferous tubules. Eosinophils were absent throughout. Atrophy of the tubular epithelium and fibrosis were noted near the periphery, under the tunica albuginea. No endarteritis was noticed.

b Epididymis — This presented an interesting picture. It was difficult to identify its structure because the region of the epididymis was occupied by a circumscribed mass of necrotic material which had undergone hyaline degeneration. But sections of degenerating adult filariæ could be identified. This area was surrounded by a zone of round-cell infiltration with some admixture of plasma and eosinophil cells and foreign-body giant cells. Finally, there were concentric rings of fibrous tissue. This picture resembles that of caseating tubercle, but no tubercle bacilli could be demonstrated. Blood-vessels did not show any endarteritis or perivascular infiltration.

c Tunica albuginea — Anterior to the body of the testicle several transverse and oblique sections of adult female filariæ (*W bancrofti*) could be seen, and numerous embryos were contained within the uterus of the parent worm. The smooth cuticle of the latter could be identified in some sections and the sheaths of the embryos in the others. The worm was placed within dilated lymphatics and in some areas it was surrounded by a well-stained zone of granulation tissue, infiltrated with eosinophil and plasma cells (*Fig 171*). But the inflammatory reactions were not seen in all sections. No male worm could be identified, although it is usually coiled up with the female worm (*Fig 172*).

d Vaginal sac — This was completely obliterated. There was a sort of fibrous capsule surrounding both the testis and the epididymis, formed by dense connective tissue (*see Fig 170*).

e Spermatic cord — The vas deferens did not show any gross lesion, but dilatation of the lymphatics was well marked.

Note — Sections were stained for bacteria in tissue by modified MacCullam's method, but no organisms were seen, neither was the *Treponema pallidum* seen in sections stained by modified Levaditi's method.

COMMENTARY

In the differential diagnosis of this case the following conditions had to be considered (1) New growth of the testicle, (2) Diffuse tertiary syphilitic orchitis or gumma, (3) Hæmatocele, and (4) Epididymo-orchitis of filarial origin.

The insidious onset, tardy rate of growth, history of venereal disease, moderately positive Wassermann reaction, and firmness in consistency of the swelling were points in favour of a diffuse syphilitic orchitis rather than a gumma. The most important points against it were the presence of testicular sensation, lymphangiectasis of the spermatic cord, and the history of two previous attacks of pain in the testicle with successive increase in size. These points were rather in favour

of a filarial complication. On the same grounds, and in the absence of any history of trauma, the possibility of the hæmatocele was excluded. The therapeutic test for syphilis was given a trial and potassium iodide was also administered in big doses. In spite of energetic antisyphilitic treatment, no improvement was noticed. Clinically, the question of malignancy in such an enlargement of the testicle could not be settled. Therefore the differential diagnosis was narrowed down to two conditions—namely, a new growth and a testicular enlargement of filarial origin.

Some of the important clinical features may be emphasized here. The testicle was adherent to the scrotum anteriorly, presenting a small fluctuating area, through which about a drachm of sterile necrotic fluid was evacuated. During the patient's stay in the hospital for four weeks, the course of the disease was apyrexial. The blood report showed an eosinophil count of 2 per cent, but no definite cause could be discovered to explain it. The blood was not examined again for the microfilaria because it was decided to remove the testicle by operation, and because, in my experience, the presence of microfilaria in the peripheral blood is a test on which much reliance cannot be placed. Brug¹ even says that "during an attack of lymphangitis all microfilariae disappear from the blood and it seems reasonable to explain this by the death of microfilariae and perhaps of the adult worm." The complement-fixation test is of value, especially after an attack of lymphangitis. Unfortunately, this test could not be done in the hospital.

SUMMARY

1 A case of an unusual and chronic enlargement of the testicle, with a very interesting clinical history, has been described.

2 The testis and epididymis, which is converted into a necrotic mass, are embedded in a connective-tissue matrix, surrounded by a thick fibrous capsule, obliterating the vaginal sac.

3 Microscopically, sections of adult female worms (*W. bancrofti*) and numerous microfilariae, contained within the uterus, are seen in the tunica albuginea. Degenerating adult filariae are also seen in the epididymis.

4 No evidence of secondary pyogenic infection could be adduced. The conclusion is reached, therefore, that the adult filaria is the real or essential cause of the pathological changes in the testicle.

5 The condition has been described under the name of chronic epididymo-orchitis or fibrosis of the testicle of filarial origin.

I wish to express my gratitude to Lieut-Colonel K. S. Thakur, I.M.S., Surgeon-Superintendent of the Howrah General Hospital, and Lieut-Colonel R. N. Chopra, I.M.S., Officiating Director of the Tropical School of Medicine, for their kindness in giving me every facility for my work. My best thanks are also due to Dr. R. Sen Gupta for his help with the histological specimens, and to Professor C. C. Bose, Dr. D. N. Banerjee, and Dr. S. Sundar Rao, for the examination of the slides and for the preparation and study of serial sections.

REFERENCE

- ¹ BRUG, S. L., "Filariasis and Elephantiasis", *Nederl. Tijds. v. Geneesk.*, 1932, June 4, 2772 (*Ext. Trop. Dis. Bull.*, 1932, Nov. 767).

TWO CASES OF OSTEOGENESIS IMPERFECTA WITH BLUE SCLEROTICS IN NATIVES OF INDIA

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THE association of osteogenesis imperfecta (idiopathic osteopsathyrosis) with blue sclerotics has long been known, and according to Jones and Lovett an hereditary factor is present in 15 per cent of cases. The writer had never met with a case during twenty-seven years service in India, nor can any reference to its occurrence in this country be found in such literature as is available here. The admission to the Medical College Hospital, Calcutta, of two children suffering from this condition within the same week, in both of which the hereditary factor in the coloration of the sclerotics could be demonstrated, seems of sufficient interest to be placed on record.

Case 1 —A Bengali boy, aged 10, admitted on Aug 28, 1933. The father was perfectly healthy in all respects. The mother was said to have been weak and sickly up to the age of 12 and to have suffered from fits of unconsciousness supposed to be epileptic in nature. These ceased as she grew up, though she was still of a neurotic temperament and suffered from palpitation of the heart. She had a miscarriage before the birth of this boy and another one subsequently, after which a second healthy boy was born. There are no other children. With some difficulty she was persuaded to come and see me at hospital, and, as I expected, she had well-marked blue coloration of the sclerotics. She was a well-developed woman, apparently in excellent health with regular features and of a very light brown complexion, the combination of which with the blue sclerotics presented (to European eyes) a very attractive picture. She brought her younger son with her and he also had the same blue coloration of the sclerotics, but had never sustained any fractures and was normal in all respects. No history of blue sclerotics or of fractures in her parents could be elicited.

The father said that the patient had always been a weakly child and had suffered from diarrhoea and irregular fever in infancy which had been diagnosed and treated as rickets. He had sustained no fewer than seven fractures between 1923 and 1927, the first at the age of three months, and all following slight violence—namely, twice of the left femur, twice of the right femur, once of the right tibia, and twice of the left forearm. The present admission was for a fracture of the right radius and ulna in the lower third. The fractures all caused only slight disability and united readily with simple splinting. From the appearances in some of the radiograms it would appear probable that several of them were greenstick fractures. The patient had lately suffered from attacks of nose bleeding. Except that the chest was rather flattened and the abdomen protuberant, the child was well developed and all the organs were healthy. There was no deformity of the skull, such as is described as occurring in severe cases, nor any deafness. In the lower third of

the right leg there was a curvature of the tibia with convexity forwards and slightly outwards at the site of an old fracture. The blood calcium was 10.5 mgrm per cent. Wassermann reaction negative.

Fig 173 shows the blue colour of the sclerotics very well, in striking contrast with the dark skin and brown irises. Radiograms were made of the whole skeleton. The skull showed a curious fine mottling of the bones of the vault, producing a



FIG 173—Case 1. Drawing showing the blue colour of the sclerotics.

spongy appearance. The right forearm showed the fracture of the radius and ulna for which the patient was admitted (*Fig 174*). The right femur showed decalcification with bowing and some attempt at buttressing on the inner side, the result of the old fracture. There was considerable thinning of the compact layer in parts unaffected by the process of union of the fracture. The left femur, which also sustained a fracture, showed similar appearances. The right leg showed a bending of the tibia and fibula which somewhat resembled the appearances seen

in the later stages of rickets, but the epiphysial lines were clean-cut, showing no signs whatever of rickets, and the bony thickenings were not confined to the concavity of the curves, but affected both sides of the shafts and were evidently due to the callus laid down at the site of a fracture, which was probably of the greenstick type. The other bones all showed general decalcification and lightness of structure, in some cases with an appearance of longitudinal striation which radiologists describe as of frequent occurrence in this disease.

The fracture was reduced under anæsthesia and put up in plaster-of-Paris. It united readily in the normal time, and the patient was discharged with a good result.

Case 2—An Anglo-Indian (Eurasian) girl, aged 10, admitted on Aug 25, 1933, with a fracture of the left femur. Her mother, who was dead, was said to have had blue sclerotics, the



FIG 175—*Case 2* Left femur

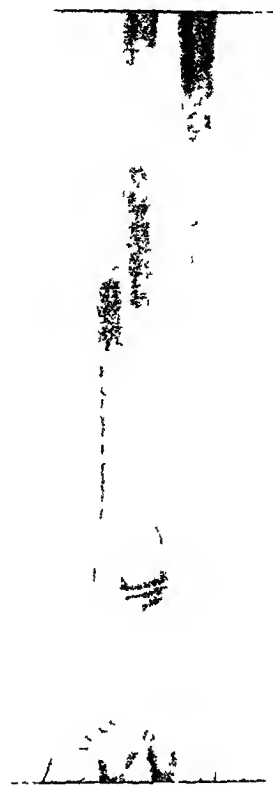


FIG 174—*Case 1* Right forearm

father I did not see, but the step-mother brought up for my inspection the younger brother and sister of the patient, aged about 8 and 9 respectively, who both had blue sclerotics, though not so marked as in this patient. Neither of these children had ever had any fractures. The history of the previous fractures was rather vague, all we could obtain was an account of a slight injury to the left ankle some months before, which was not recognized as a fracture, though the radiograms which we took clearly showed a recently united fracture.

The child was healthy in all respects and well developed. The blood calcium was 10 mgrm per cent.

The radiogram of the injury (*Fig 175*) showed a spiral fracture in the middle third of the left femur. This was treated by Hamilton Russell's method of extension, and *Fig 176* shows the result six weeks later—good callus formation in perfect position and an excellent clinical result, as is usual in these cases.

The other radiograms showed the same general decalcification and lightness of the compact bone as in *Case 1*, but not to such a marked degree. *Fig 177* obviously shows a recently united greenstick fracture of the left tibia, though the history records only a sprain, and the curvature of the bone was evident clinically. In the right leg there was a thickening in the lower third of the fibula and a slight inward bend of the tibia, evident in the radiogram though not clinically, probably due to weight-bearing during the healing of an undiagnosed fracture. The skull showed a mild degree of the same fine mottling observed in *Case 1*.

DISCUSSION

Both the cases appear to be examples of the more common type of the disease, seen in older children and adolescents, with thinned out cortex, but no special alteration in internal texture of the bones, though *Case 1* showed in the skull the honeycomb structure associated with the more severe type, in which general softening of the bones leads to bending rather than breaking, but the rest of the

FIG 176—*Case 2*. Left femur six weeks after treatment

skeleton showed only a slight indication of this condition in the shape of a longitudinal striation of some of the long bones, and the radiograms showed that the curvatures of the bones were all due to old fractures. There was no question of rickets playing any part in the causation in either case, as the characteristic radiological changes of that disease were entirely absent and the epiphyses in both cases were perfectly normal.

There is a tendency in some cases for the skeleton to become stronger and the fractures less frequent after puberty, and it appears likely that such a mild case as *Case 2* will outgrow the disease, but *Case 1* was a more severe type and in this patient the prognosis is not good.

Both these cases were sent to Lieut-Colonel Kirwan, F.R.C.S.I., I.M.S.,

FIG 177—*Case 2*. Left tibia and fibula

Professor of Ophthalmology, Medical College, Calcutta, for his opinion on the eye condition, and a note by him on the subject of blue sclerotics is appended

"Although many cases of blue sclerotics have been reported in ophthalmic literature, the condition is uncommon in India, and during the six and a half years I have been Professor of Ophthalmology in the Medical College, Calcutta, I have seen only two cases—one was in an Anglo-Indian girl and the other in a Bengali boy

"Congenital blue sclerotics are characterized by abnormally thin scleras extending from the corneal margin as far back as can be seen, and are accompanied by a failure in the development of the connective tissues. This condition is frequently accompanied by fractures of the long bones, dislocations and sprains of the joints, deafness due to otosclerosis, small stature, and dental affections. The thinness of the sclerotics is hereditary in nature and it is usually possible to get histories of its occurrence in several successive generations. It is transmitted by either male or female through affected persons only, and is present, according to Kunii,¹ in half of the relatives

"The etiology of the condition is still open to doubt. Two theories are brought forward—one that it is due to an increased transparency of the sclera owing to the lack of fibrous tissue and lime salts, and the other that it is due to an increase in the pigment in the cells of the sclera. Some others consider that it is due to poverty of the mesodermal layers, whilst others dispute this hypothesis, as often ectodermal structures are affected and blue scleras have been found in many congenital defects of the eye both of mesodermal and ectodermal origin."

REFERENCE

¹ KUNII, *Zeits f Augenheilk*, 1930, July, 328

CHOLECYSTITIS WITHOUT STONE

AN INVESTIGATION OF 264 OPERATED CASES FROM THE CLINICAL, RADIOLOGICAL, AND PATHOLOGICAL ASPECTS AN ATTEMPT TO DETERMINE THE FACTORS OF SERVICE IN ESTIMATING PROGNOSIS *

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SYNOPSIS

INTRODUCTION

Historical note

Is prognosis after cholecystectomy better in cholelithiasis or in cholecystitis without stone?

Is the case-history or the histology of the gall-bladder the more reliable guide in estimating prognosis after cholecystectomy?

THE PRESENT INVESTIGATION (264 CASES)

Method of selection of cases

Survey of pathological material

The clinical results after cholecystectomy

Statistical analyses

- 1 The relation of local conditions around gall-bladder to clinical end-result
- 2 The relation of the pre-operative history to the clinical end-result
- 3 The relation of microscopic pathology to the clinical end-result
- 4 The relation of cholecystographic findings to pathological changes in the gall-bladder
- 5 The relation of cholecystographic findings to the clinical end-result

GENERAL CONCLUSION

INTRODUCTION

It is only fifty-two years since Langenbuch performed the first cholecystectomy (1882), thus introducing an operation which has become one of the most frequent in surgery. Its widespread application is an even more recent development, and great changes in the indications for it have taken place even in the last few years. The early practice of gall-bladder surgery is exemplified by a series of 12,000 cholecystectomies by German surgeons collected and published in 1923 by Hotz. The figures referred to a period when cholecystectomy was a definitely hazardous procedure, and so the principal point of interest was the mortality rate, which averaged 10 per cent. When ultimate prognosis was considered, recrudescence of pain was the usual expectation in 25 per cent of cases and minor complaints were lightly regarded. Hotz felt this to be an unsatisfactory state of affairs, and largely attributable to the respect accorded to the teaching of Kehr (1913)—that symptoms

* This work was done during tenure of a Rockefeller Foundation Travelling Fellowship in Surgery, 1932-3

will subside spontaneously in 80 per cent of gall-stone cases, and accordingly operation should be reserved for the remaining 20 per cent, in all of these it should be regarded as a necessary and life-saving measure, but the others should not be subjected needlessly to the risk, even though slight, of a gratuitous operation. Hotz challenged these conservative views, and advocated operation immediately the diagnosis of gall-stones had been made, for, in the relatively young, without degenerative changes in vital organs, operation would carry little risk (mortality about 4 per cent), and a good ultimate prognosis could be given. On the other hand, in cases where the disease had been allowed to drag on to old age, risks were so high, and recrudescence of symptoms so frequent, that one would hesitate to interfere.

It will be noted that in the period covered by Hotz's review biliary surgery was practically limited to cases of gall-stones, but since that day its scope has widened greatly. The introduction of cholecystography by Graham and Cole in 1924 opened new avenues of approach to the study of biliary physiology and pathology, stimulated interest in gall-bladder surgery, and led undoubtedly to wide extension of its practice, for 'cholecystitis' was diagnosed at an earlier stage, and many gall-bladders were removed that previously might have been left *in situ*. In 1927 Judd from his wide experience stated that "The number of operations upon the gall-bladder has been greatly increased since the general employment of the Graham-Cole method in making the diagnosis." In the following year Hirsch and Taylor proposed the admonitory query, "Are we approaching an era similar to that happily passing in reference to the appendix, when the gall-bladder is to be removed on slight pretext?" This important question can be answered only by critical analysis of the end-results in a great number of cases subjected to cholecystectomy, and it is part of the object of this paper to contribute some information of this kind.

Much of the increase in the employment of cholecystectomy may, of course, be attributed to reduction in operative mortality. The decision to perform any operation of election is made only when it is estimated that the probability of ultimate benefit sufficiently outweighs the operative risk. Operative mortality in biliary surgery has been enormously reduced in recent years through the application of new knowledge of liver physiology, and, as an aid in assessing the risks of operation, the use of tests of liver function (e.g., Graham, Cole, et al., 1926), correlated with appropriate pre-operative treatment. A reasonable degree of integrity of the metabolic and detoxifying activities of the liver is essential for the safe performance of any major operation, especially under general anaesthesia, but since disease of the gall-bladder shows an especial tendency to lead to destructive hepatic lesions, it is all the more necessary in this branch of surgery to determine, before embarking upon an operation of election, that the functional capacity of the liver is satisfactory. By the routine use of these principles and methods, Graham has reduced his operative mortality from 6 per cent to the extraordinarily low figure of 0.4 per cent (1931).

The risk of attack upon the gall-bladder, therefore, has been to a great extent eliminated, and interest has tended to move away from mortality statistics, bacteriology, and pathology, and to be focused upon end-results and theories of gall-bladder function, regarding all of which there has been most voluminous but extraordinarily conflicting writing. Thus writers are far from agreement upon the

apparently simple question of *whether the prognosis after cholecystectomy is better in cholecystitis without stone or in frank cholelithiasis* Hartmann and Petit-Dutailis (1922), Alvarez (1923), Deaver (1925), Zink (1926), Deaver and Bortz (1927), and Connell (1928) have all published their findings that results are better in the former condition, and explain these by the assumption that cholecystitis without stone represents the earliest stage of gall-bladder disease and that when the stage of cholelithiasis is reached there have developed, in a large proportion of cases, secondary lesions in other viscera, which persist after cholecystectomy and maintain symptoms. On the other hand, Florcken (1923), Gundermann (1924), Seuleberger (1925), Hitzrot and Cornell (1926), Muller (1926), Lahcy (1927), Young (1928), Schondube (1928), Feinblatt (1928), Ross (1932), and Stanton (1932) all found that the clinical results are better in cholelithiasis, and argue that many stoneless gall-bladders removed at operation have not in fact been the cause of the symptoms with which they have been labelled.

It would be strange were there no middle position in this moot question, and indeed there is—namely, that adopted by Hueck (1928) and by Judd and Priestley (1932), who could find no significant difference in the results achieved by cholecystectomy in cholelithiasis and in cholecystitis without stone.

In an attempt to discover with which of the large opposing schools the balance of truth may lie, an analysis has been made of practically all of the statistics of the end-results after simple cholecystectomy published during the last ten years, representing rather over six thousand cholecystectomies of wide geographic distribution.

The evidence obtained may be summarized by saying that “*The experience of a large number of surgeons in the past has shown that cure or improvement after cholecystectomy may be expected in nearly 90 per cent of cases of cholelithiasis and in rather over 80 per cent of cases of cholecystitis without stone*”

Is Greater Reliance to be Placed upon the Case History or upon Pathological Examination in Assessing Prognosis? It is interesting to study the recoil in recent years from the pathological point of view toward the clinical in gall-bladder diagnosis.

Criticizing Alvarez' paper in 1923, MacCarty stated definitely, “I do not believe in removing a gall-bladder on a history”, while in 1924, drawing a parallel with the operation of gastro-enterostomy, Judd and Burden began an article with the dictum, “Removal of the gall-bladder on the basis of symptoms and clinical history in the absence of a local lesion invites discredit on the judgment of the surgeon and on the operation”. In the following year Judd stated, “Operations based on clinical symptoms alone are likely to fail”. In the same place, however, when discussing the stoneless gall-bladder he said, “In the presence of colic the diagnosis is certain, but much uncertainty attends attempts to diagnose chronic cholecystitis in the absence of colicky pain. As a general rule operative procedures must be based on a tangible lesion found at operation and not on physiologic changes or clinical symptoms”. Finally, in 1927, Judd formulated his opinion as follows: “While one hesitates to remove a gall-bladder in the absence of definite pathology, if the gall-bladder is not removed in these clinically definite cases, symptoms will continue. If it is removed the pathologist generally finds the changes of chronic cholecystitis”. This last fact loses some of its importance, however, when it is remembered that Carman, MacCarty, and (1924) found only al

gall-bladders in a consecutive unselected series of 5000 obtained at autopsy. Clearly it is unlikely that more than a small proportion of these 'abnormal' gall-bladders had given rise to trouble during life, and the clinical value of emphasizing such slight changes in the histology of the gall-bladder would seem therefore to be doubtful. Schmieden (1921) published an account of his views on dyskinesia of the biliary tract, and stated that in one year he had removed fourteen stoneless gall-bladders, with cure of symptoms of biliary colic in all. He concluded that, even in the absence of pathological changes, provided always that symptoms were severe, cholecystectomy would achieve good results in these cases of dyskinesia, this is the direct reverse of the standpoint, quoted above, adopted by Schondube. Deaver and Bortz (1927) concluded that the most important factor in making a diagnosis of gall-bladder disease is the clinical history. In the same year Lahey stated, "If operation is done for a definite chain of symptoms unrelieved by medical treatment, then the normal appearance of the gall-bladder would not deter one from removing it, for it may be a cholesterol gall-bladder."

Young (1928), reviewing a large series in which the diagnosis was made before cholecystography was available, found that, generally speaking, those cases in which there is not a definite history of biliary colic are the least likely to be improved by cholecystectomy. "The only justification for operation on the biliary tract should be well-established clinical and laboratory evidence that the gall-bladder is at fault, and having made that diagnosis, it should be removed whether or not it seems diseased to the examining hand." Very recently Stanton (1932) has re-asserted his conviction of 1927 that "The only reliable guide is the presence of attacks of definite gall-bladder colic, and if the surgeon is sure of such a history, he can confidently remove the gall-bladder even in the absence of gross pathology."

There is a further group of symptoms commonly ascribed to gall-bladder disease and occasionally referred to in surgical text-books as those of the pre-calculous stage of cholecystitis—flatulent dyspepsia, food-selection (inability to tolerate certain articles of food, especially fats, certain vegetables such as cabbage, or raw fruits), and constipation. Some doubt exists, however, regarding the diagnostic significance of these symptoms.

It is clear from the above review that the majority of surgeons have come to regard biliary colic as a symptom which definitely implicates the gall-bladder, although the condition may be functional and not organic. Further, many hold the opinion that, if colic has occurred, a satisfactory outcome from cholecystectomy may be practically guaranteed. In fact, in estimating prognosis, this symptom is considered to be of greater importance than the pathological changes that may be present in the gall-bladder.

A smaller number of writers have attributed an almost equal significance to the less prominent symptoms—flatulent dyspepsia, food selection, and constipation—which go to complete the syndrome regarded as characteristic of gall-bladder disease.

It would be possible to continue this balancing of conflicting beliefs and published statements regarding practically every important point in biliary surgery, but enough has been said to show that there is still much need for detailed investigation and accurate observation.

THE PRESENT INVESTIGATION

This investigation was undertaken at the kind suggestion of Dr Evarts A Graham, Professor of Surgery at Washington University Medical School, St Louis, Mo, U S A Its principal object is to discover the end-results after cholecystectomy in cases of cholecystitis without stone

METHOD OF SELECTION OF CASES

The material for the survey consists of the cases in which, during the ten-year period 1922-31, cholecystectomy was performed by the surgeons on the staff of Barnes Hospital, St Louis The investigation concerned all cases of cholecystitis without stone, and all cases of cholesterosis of the gall-bladder The cases in which cholesterosis was complicated by the presence of stone were also included

During the period in question appendectomy was performed routinely at the same time as the cholecystectomy, and a small piece of liver removed for microscopic examination (biopsy), but no case has been considered in which there was a more extensive surgical procedure, or in which the stoneless condition of the gall-bladder was due apparently to a previous cholecystostomy The adoption of these uniform standards of treatment makes possible a fairly accurate analysis of end-results in their relation to variations in clinical history, microscopic findings, and radiological data

A letter of inquiry was sent to each patient, and in cases where the family physician was known, his opinion in addition was sought The letter to the patients contained six questions —

1 Do you consider yourself cured, improved, or unimproved by your operation ?

2 Do you have any indigestion now ?

3 If so, is this indigestion of the same character as that which you had before ?

4 If not, will you state your principal complaints, such as pain, bloating, nausea or vomiting, constipation, belching, jaundice, sour mouth ?

5 Is it necessary for you to omit certain kinds of food from your meals ?

6 If so, what kinds—fried foods, uncooked fruits and vegetables, etc ?

To the majority of those expressing themselves as unimproved, an invitation was sent to come for examination, and in a few it was found that definite improvement had in point of fact been achieved, but in the greater number the replies to the questionnaire proved reliable

SURVEY OF PATHOLOGICAL MATERIAL

In order to arrive at a uniform assessment of the gall-bladder lesions under consideration, the pathological material, all of which is preserved in the Department of Surgical Pathology, Washington University, was reviewed The following brief paragraphs are introduced in order to define accurately the terms of classification The number of accompanying photomicrographs has been reduced to a minimum and these represent merely a few examples illustrative of the conditions described

INTERPRETATION OF THE TERMS OF PATHOLOGICAL CLASSIFICATION

The Gall-bladder with Minimal Lesion (*Figs 178, 179*)—The gross specimen is relatively thin-walled and contains well-coloured clear bile. Microscopically, the mucosa is plicated to a degree depending upon the state of distension and the site of origin of the specimen (the mucosal folds being taller near the neck). There is often a considerable amount of lysis of the columnar epithelium, due to action of the bile-salts after ligation of the cystic artery. The sub-epithelial layer consists of rather dense poorly cellular fibrous tissue, forming a delicate core for each fold. This layer contains a few lymphocytes. The muscularis is a thin sheet of interlacing strands of fine smooth fibres, rather intensely eosinophil. It is rare for the muscular tunic to be penetrated by pouchings of the mucosa, but on occasion one finds a pocket of the latter escaping through an hiatus in the muscularis, and forming a dilated vesicle in the subserous coat, without any accompanying evidence of recent or old inflammation. The amount of subserous fibrous and elastic tissue varies,

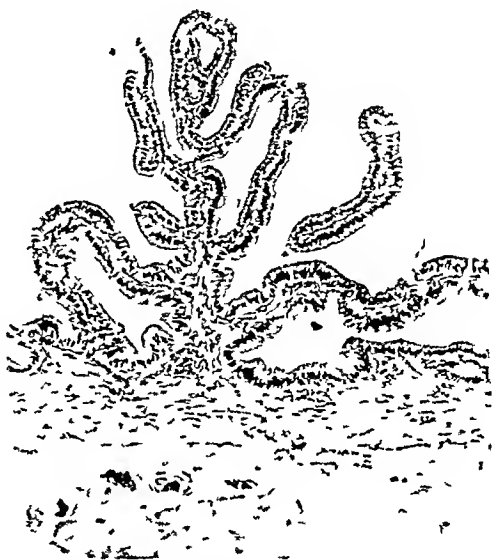


FIG 178—Minimal lesion. Specimen from body of gall-bladder. Complex plicate arrangement of mucosa. Plica shows extremely delicate core of mature vascular fibrous tissue with moderate lymphocytic infiltration. Thin well-defined muscular layer ($\times 50$)



FIG 179—Minimal lesion. Almost complete lysis of lining epithelium due to slight delay in fixation. In the presence, however, of the thin muscular coat, delicate plical cores, and normal subserous layer all free of inflammatory infiltration, this extreme lysis is merely additional evidence that in life there has been little departure from normal in the gall-bladder or in its content ($\times 50$)

but one should be able to distinguish separate wavy bundles of mature collagen fibres. A tendency to dense lamination is evidence of previous definite inflammatory episodes, after which it is rare for restitution to be so complete that the gall-bladder lesion may be termed 'minimal'.

Chronic Catarrhal Cholecystitis (*Figs 180, 181*)—The most prominent characteristic of this condition is mucosal œdema, with clubbing of the tips of the plicæ. In addition, lymphocytic infiltration is increased, not only in the mucosa but also in the outer coats. The epithelium is better preserved than in gall-bladders more nearly normal, because of



FIG 180—Chronic catarrhal cholecystitis. Typical specimen. Marked oedema with clubbing of ends of plicae. Some muscular thickening and increase of fibro cellular tissue of mucosa ($\times 50$)



FIG 181—Chronic catarrhal cholecystitis. Advanced case with great muscular thickening and formation of muciniferous glands of gastric type. The lysis of the superficial epithelium is an artefact ($\times 50$)



FIG 182—Chronic fibrous cholecystitis. Enormous thickening and hyaline change in muscular layer. Mucosal plicae more or less absent. Epithelium closely applied to muscle bundles, between which crypts herniate ($\times 50$)



FIG 183—Chronic fibrous cholecystitis. Extreme crypt formation. Crypts around artefact ($\times 50$)

course the bile is less concentrated. The epithelial cells tend towards cubical type, and muciniferous elements may be increased in number either by multiplication of the goblet-cells normally present or by new-formation of actual mucous glands in parts other than the neck, where they are normally present. To these abnormal glandular structures the term 'gastric glands' is sometimes applied. The muscular coat is moderately thickened. Mucosal crypts penetrating the muscular coat are slightly more common than in gall-bladders showing minimal lesions.

Chronic Fibrous Cholecystitis (*Figs 182, 183*)—It is rare for the stoneless gall-bladder to be the seat of this advanced lesion, which develops usually as a result of repeated attacks of obstruction by stone. The mucosa is degenerate, with cubical epithelium set upon squat plicæ of rather dense fibrous tissue into which muscular bundles often project. The close application of the epithelium to the muscularis, and the marked tendency for the formation of diverticular epithelial crypts extending completely through the muscular coat, are among the prominent features of the condition. When fully developed the crypts form large dilated simple glandular pockets, surrounded by a thick layer of new-formed fibrous tissue which shows a degree of inflammatory infiltration dependent no doubt upon the presence or absence of infection, and the time which has elapsed since the last obstructive attack. In the intervening areas the muscular layer is greatly thickened, owing in part to a sort of hyaline swelling of the individual fibres, which in section may appear like empty shells, and in part possibly to actual increase in their number. The subserous coat shares in the general thickening and often there is marked endarteritis of the larger vessels.

Cholesterosis of the Gall-bladder (Strawberry Gall-bladder) (*Figs 184-186*)—These are the names given to a very common condition in which the gall-bladder mucosa shows a yellow stippling owing to lipid deposits situated principally at the tips of the plicæ. Its frequency may be appreciated from the fact that Mentzer (1925) found some degree of cholesterosis of the gall-bladder in 38 per cent of all cases coming to autopsy at the Mayo Clinic. Regarding its ultimate nature, however, there is on many points the most acute divergence of opinion. Accordingly it will be considered in detail in a later paper, and meanwhile only a brief description will be given. Essentially it consists in the accumulation of doubly refracting cholesterol esters and neutral fats principally in two sites—namely, in the lining epithelium and in macrophages in the connective tissue of the mucosa. The deposit is patchy and forms bright yellow flecks visible to



FIG 184—Cholesterosis. Massive lipid deposit confined to the lining epithelial cells. In some of these the lipid is so abundant that they have taken on the histological character of foam-cells. Some lymphocytes in all coats, and possibly slight muscular thickening, but no unequivocal inflammatory signs ($\times 50$)

the naked eye, sometimes slender and scanty, sometimes filling each mucosal fold so that the gall-bladder appears to be lined with a thick soft fabric woven of coarse golden waxy threads. Quite frequently yellow morular polypi hang by slender stems, and in some cases these polypi may be the sole sites of gross lipid deposit. The subserous coat often contains an unusual amount of fat. In about a third



FIG 185—Cholesterosis. Interstitial cholesterosis and pedunculation in a gall bladder showing no inflammatory signs whatever ($\times 50$)



FIG 186—Cholesterosis. Lipid deposit in this section confined to a single stromal cell of a gall bladder showing most extreme degree of inflammatory infiltration and thickening ($\times 50$)

of the cases of cholesterosis, gall-stones are found, and they are almost always of cholesterol-rich type—cholesterol solitaires and mulberry stones.

During the decade under review, cholecystectomy was performed on 262 cases which may be classified conveniently into five categories of gall-bladder lesions

Table I—NUMERICAL DISTRIBUTION OF LESIONS UNDER CONSIDERATION
PATHOLOGICAL MATERIAL AVAILABLE IN 262 CASES

	TRACED	UNTRACED	TOTAL
Minimal lesion	57	46	103
Cholesterosis	33	13	46
Chronic catarrhal cholecystitis	53	30	83
Chronic fibrous cholecystitis	5	4	9
Cholesterosis with stone	18	3	21
TOTALS	166	96	262

In two additional cases the pathological material was not available. The number of cases in the various pathological categories are given in Table I, which shows also that it has been possible to trace the clinical outcome in 166

It will be seen that, on histological grounds, rather more than a third of the cases have been termed 'minimal lesion'. This demonstrates that the present series embraces a considerable proportion of cases of quite 'early' gall-bladder disease. It is fairly certain that, had a specific search for cases of cholesterosis been made in the early years, more would have been found. In checking the pathological material a few cases were discovered which previously had been overlooked, but, generally speaking, inspection of the fresh specimen will discover degrees of lipid infiltration difficult to identify histologically without special methods.

THE CLINICAL CONDITION AFTER CHOLECYSTECTOMY FOR CHOLECYSTITIS WITHOUT STONE

Of a total of 264 cases suited for this study it was possible to examine the pathological material and also to ascertain the end-result in 166, 148 stoneless, and 18 cases of cholesterosis with stone. In assessing the post-operative state of a case as *well*, *improved*, or *unimproved*, the opinion of the patient on the matter has been the most important criterion. Now, if a patient has been subject to attacks of biliary colic, he will be delighted to be relieved of this, and will happily bear such minor distresses as flatulence and avoid certain foods which he finds are followed by discomfort. On the other hand, if the original symptoms have not been so severe, slighter residua will be a source of complaint. Thus it comes about that certain of the patients classed as unimproved have possibly no greater disability than have some who are designated 'well', yet this subjective method of classification appeared to be the most satisfactory.

Of the 149 cases of cholecystitis without stone, 44 (29.2 per cent) were classed as cured, 44 (29.2 per cent) as improved, and 53 (36.2 per cent) as unimproved. There were 8 deaths.

The series of 18 cases of cholesterosis complicated by the presence of stone is of course small, and cannot be regarded as an adequate control, in comparison with the stoneless cases, yet it is worthy of mention that none failed to be improved.

MORTALITY AFTER CHOLECYSTECTOMY FOR CHOLECYSTITIS WITHOUT STONE

That there should have been 8 deaths in this series of stoneless gall-bladder cases is surprising. Although *Table V* indicates an operative mortality of 5.4 per cent, this is misleading, for the mortality should of course be calculated on all the operations and not merely on the number of cases subsequently traced. The mortality so adjusted is 3.3 per cent, a low figure, but not lower than the average mortality for gall-bladder operations of every kind during the period, so that the absence of advanced pathological changes in a gall-bladder is not in itself evidence that cholecystectomy will be a procedure free from risk. It is clear that when there is little amiss with the gall-bladder, there is a greater chance that diagnosis may not have been accurate and that the symptoms may have been due to some other unsuspected cause, under which circumstances the operation can do little but harm. There is the further possibility that removal of a fairly healthy gall-bladder, which still retains a proportion of its normal function, may produce more violent systemic derangement, and hence be more dangerous, than removal of an effete organ.

The pathological condition of the gall-bladder in the 8 fatal cases was as follows Minimal lesion 3, cholesterosis 1, chronic catarrhal cholecystitis 4

From this preliminary survey, it has emerged that in cases of cholecystitis without stone treated by cholecystectomy we may expect an operative mortality of about 3 per cent, cure of symptoms in 30 per cent, improvement in 30 per cent, and an unsatisfactory result in 37 per cent

It is clearly desirable that the operation of cholecystectomy should be withheld in this last group, and the remainder of this paper consists largely of various analyses of the results to determine if possible the characteristic features of cases favourable for cholecystectomy

STATISTICAL ANALYSES OF CLINICAL, PATHOLOGICAL, AND RADIOLOGICAL INFORMATION, AND CORRELATION WITH ULTIMATE OUTCOME

LOCAL CONDITIONS FOUND AT OPERATION IN RELATION TO PROGNOSIS

It was found that the cases of cholecystitis without stone could be divided on the basis of local appearances into two large groups, namely, those in which adhesions were limited to the neck and infundibulum of the gall-bladder, attaching it to the first part of the duodenum (*Series A*), and those in which the gall-bladder was more or less buried in adhesions (*Series B*) *Table II* shows the clinical end-results in these two series

Table II—THE RELATION OF LOCAL PATHOLOGY TO LATE CLINICAL RESULT AFTER CHOLECYSTECTOMY 149 CASES OF CHOLECYSTITIS WITHOUT STONE

TYPE OF CASE	TOTAL	WELL	IMPROVED	UNIMPROVED	POST OPERATIVE DEATH
<i>A</i> Cases with adhesions limited to region of neck of gall-bladder	72	22 (31%)	12 (17%)	33 (45%)	5 (7%)
<i>B</i> Cases with extensive adhesions more or less covering gall-bladder	77	22 (29%)	32 (41%)	20 (26%)	3 (4%)

It will be seen that of *Series A* approximately one half were cured or partially relieved of their symptoms, and of *Series B* 70 per cent. The majority of the poor results occurred in those cases with only slight signs of previous inflammation around the gall-bladder. Of these only half derived any benefit from the operation. It would be most valuable, however, were it possible to determine *before operation*—e.g., from study of the history—which case is likely to do well, and which to give a poor result. In an attempt to secure information which may be used in this way an analysis has been made in which the types of pre-operative symptoms are correlated with the end-results after cholecystectomy (*Table III*)

CLINICAL HISTORY AND ITS RELATION TO PROGNOSIS

As already mentioned, many authors maintain that cases with a history of biliary colic are practically certain to do well after operation while cases without such a history are likely to do badly. This statement is readily tested in the present series since the presence or absence of biliary colic makes a very clear-cut

division between two types Biliary colic has been taken to mean pain of considerable severity, originating in the epigastrium and radiating backward, usually to the angle of the right scapula, although it is known that biliary pain does not always conform to this description

Table III—CORRELATION OF BILIARY COLIC AND PATHOLOGICAL CHANGES WITH END-RESULTS AFTER CHOLECYSTECTOMY IN 140 CASES OF CHOLECYSTITIS WITHOUT STONE

PATHOLOGICAL LESION PRESENT	47 CASES WITH HISTORY OF BILIARY COLIC			93 CASES WITHOUT HISTORY OF BILIARY COLIC		
	Well	Improved	Unimproved	Well	Improved	Unimproved
Minimal lesion	2	6	7	9	15	15
Cholesterosis	8	0	5	6	2	11
Chronic catarrhal cholecystitis	10	6	1	7	12	13
Chronic fibrous cholecystitis	1	1	0	1	2	0
Total of stoneless cases	21	13	13	23	31	39
Percentage well or improved	76			58		

Of the stoneless cases, 47 (33 per cent) gave a history of colic, and of these 76 per cent were cured or improved by operation, while of the 93 in whom colic had not occurred, only 58 per cent had derived benefit from the cholecystectomy. The prognosis, therefore, is distinctly better in the cases in which colic has occurred, though the difference between the two types is not extreme. The fact that a third of the cases of cholecystitis without stone had suffered from biliary colic is important, for it proves beyond doubt that this symptom is not always due to the presence of biliary calculi. On the other hand, it is probable that in the great majority of cases the symptoms of biliary colic arise as a result of obstruction of the outlet from the gall-bladder to the cystic duct. Clearly, therefore, whatever the ultimate basis of the symptoms, the latter are likely to be relieved by removal of the gall-bladder.

Conclusions derived from a more detailed analysis on the basis of the pathological subdivisions would necessitate much larger numbers. Accordingly the large percentage of improvement registered in those cases of cholesterosis and chronic catarrhal cholecystitis with a history of colic is merely pointed out without comment. Nevertheless it is interesting to recall the title of Moynihan's original article (1909) "A Disease of the Gall-bladder requiring Cholecystectomy." The six cases described came to operation for the relief of severe biliary pain, and the present series might easily have furnished a comparable group of six consecutive successes. The conclusion might then justifiably have been drawn that the symptoms of cholesterosis are often benefited by cholecystectomy, though to term it a lesion which requires cholecystectomy would possibly be unduly dogmatic.

Next to biliary colic, flatulent dyspepsia and food selection are the syndrome most generally held to incriminate the gall-bladder. Some authors are of the opinion that "it cannot be too strongly stated" that this is so, and that accordingly patients complaining of these symptoms will best be treated by cholecystectomy. This thesis can readily be submitted to the therapeutic test in the present series. Of the 167 cases which have been traced, 80 complain still of these symptoms

Only 14 are known definitely to have been relieved by the operation, and a considerable number apparently have developed flatulence and food selection during the period since the operation. This does not, of course, indicate that the operation was responsible for the appearance of these minor symptoms, for it may well be that the mere passage of the years has brought upon some these gassy upsets and distaste for certain foods. The list of foods which prove harmful varies, but generally comprises fat, pork, fried food, raw fruit, and the wind-forming vegetables—cabbage, asparagus, and the legumes.

These findings emphasize the fact already stated that, in the present series, relief of pain was not invariably accompanied by alleviation of associated minor discomforts. It seems very doubtful, therefore, whether these symptoms of flatulent dyspepsia and food selection have their origin directly in the gall-bladder. It is more probable that they arise in some disturbance of motor function elsewhere in the alimentary tube, possibly at the pylorus or in the colon. They are unlikely to be relieved by cholecystectomy.

MICROSCOPIC PATHOLOGY AND ITS RELATION TO PROGNOSIS

From the surgical aspect, value attaches to separation of the various types of lesion which affect an organ only if it is possible thereby to foretell accurately the outcome of treatment—in other words, a correlation must be made between pathological change and prognosis, and this, in the case of the gall-bladder, has never been done. It is a natural assumption that, other things being equal, the more advanced the lesion present in such an organ, the more marked will the resulting symptoms be, and the greater the relief after its removal. The preliminary discussion, however, showed that many authorities in the field of biliary surgery believe that, with the advance of the morbid process in the gall-bladder, there develop almost constantly in other organs associated lesions which cause the outlook to be less favourable, accordingly they advocate operation early in cholecystitis, before these secondary changes have had time to occur. This implies that, if the gall-bladder is removed at a stage when pathological changes in its wall are at a minimum, results will be better than when the gall-bladder is markedly diseased.

Table IV—RESULTS OF CHOLECYSTECTOMY IN 148 STONELESS CASES AND 19 CASES OF CHOLESTEROSIS WITH STONE

	TOTAL	WELL	IMPROVED	UNIMPROVED	POST- OPERATIVE DEATH
Minimal lesion	57	11	22	21	3
Cholesterosis	33	14	2	16	1
Chronic catarrhal cholecystitis	53	18	17	14	4
Chronic fibrous cholecystitis	5	2	3	0	0
Cholesterosis with stone	19	6	13	0	0

It will be seen from *Table IV* that in the series of cases in which the gall-bladder was the seat of a minimal lesion, 58 per cent derived some benefit from the operation, though only a small proportion were relieved of all their complaints. Of the cases of cholesterosis, only half were benefited, but of the latter almost all professed to be completely cured. Of the cases classified as chronic catarrhal

cholecystitis, 67 per cent secured some benefit, and in the two final categories of more definitely diseased gall-bladders, none failed to be improved, though a fairly considerable proportion continued to have some complaints, generally flatulent dyspepsia, food selection, and constipation. The cases of cholesterosis with stone have been included in the table to serve as a control upon cases less definitely pathological.

TO WHAT EXTENT MAY IMPROVEMENT BE ATTRIBUTED TO THE ASSOCIATED APPENDECTOMY?

Cases with only very slight pathological changes in the gall-bladder were improved after cholecystectomy to the number of 58 per cent. In evaluating such slight changes it is essential to determine how far the associated appendectomy was responsible for the benefit. As with the gall-bladder, so with the appendix, none is normal if importance is attached to minutiae, but it has come to be believed that it is rare for an appendix to produce marked symptoms without the occurrence of obstructive or inflammatory attacks which leave behind very definite evidence in the form of well-marked thickening, particularly of the submucosa. *Table V* shows the distribution of the cases in which definite appendicitis was present, and also the clinical result of operative treatment upon the various categories of cholecystitis. The first numeral in each column represents the total number of cases in the category, the number in brackets indicates how many of these showed definite appendicitis.

Table V—ASSOCIATION OF MARKED CHRONIC APPENDICITIS WITH GALL-BLADDER LESIONS

STATE OF GALL-BLADDER	CONDITION FOLLOWING OPERATION		
	Well	Improved	Unimproved
Minimal lesion	11 (2)	22 (5)	21 (8)
Cholesterosis	14 (5)	2 (1)	16 (3)
Chronic catarrhal cholecystitis	18 (4)	17 (5)	14 (3)
Chronic fibrous cholecystitis	2 (0)	3 (0)	0 (0)
Cholesterosis with stone	6 (0)	12 (0)	0 (0)

The surprising fact emerges that the proportion of diseased appendices is not lower but even rather higher among the unimproved cases than in those who derived benefit from the combined operation. Therefore it seems fair to conclude that the appendectomy had little to do with the outcome in most cases. If, however, those cases with a favourable result are excluded in which a definitely pathological appendix was removed, the figures in *Table VI* are obtained. These must represent the cases in which post-operative improvement was definitely due to removal of the gall-bladder. Since no exclusion was made from the 'unimproved' column, this system of evaluation must be regarded as stringently critical.

These carefully controlled observations should enable one to determine to what extent cholecystitis of the various types is specifically responsible for symptoms which are likely to be relieved by cholecystectomy—in other words, one should now be able to judge to what extent the various lesions are clinically significant. Actually the figures show that following cholecystectomy, either cure or

partial relief from symptoms resulted in 40 per cent of cases of simple cholesterosis, 52 per cent of those in which the gall-bladder showed a 'minimal lesion', and 60 per cent of those showing chronic catarrhal cholecystitis. The benefit in all of these cases may fairly be attributed to the cholecystectomy.

Table VI—GALL-BLADDER PATHOLOGY AND PROGNOSIS AFTER DEDUCTION OF CASES IN WHICH BENEFIT MIGHT BE ATTRIBUTED TO APPENDECTOMY, LEAVING 125 STONELESS AND 18 STONE CASES—143 CASES IN ALL

	TOTAL	WELL	IMPROVED	UNIMPROVED	POST- OPERATIVE DEATH
Minimal lesion	50	9	17	21	3
Cholesterosis	27	9	1	16	1
Chronic catarrhal cholecystitis	44	14	12	14	4
Chronic fibrous cholecystitis	4	2	2	0	0
Cholesterosis with stone	18	6	12	0	0

The poor results in the cases of simple cholesterosis are particularly striking, and these are worse even than in the cases with a minimal lesion. The conclusion would seem justified that so far as the production of symptoms is concerned, cholesterosis of the gall-bladder is of no significance whatever. Since only half of the cases with 'minimal lesion' derived benefit from the cholecystectomy, it is clear that this type of cholecystitis is not invariably symptom-producing. The outlook is a little better in chronic catarrhal cholecystitis, but is still not entirely satisfactory. The results indicate that undue significance should not be attached to microscopic changes of this moderate degree. In the cases with more advanced gall-bladder disease—chronic fibrous cholecystitis, and cholesterosis with stone—the results were almost uniformly good, and were comparable with the large mass of statistics of many authors mentioned earlier in this paper.

It is clear that, just as cases with well-marked symptoms are likely to do better after cholecystectomy than those in whom the symptoms are vague, so removal of a definitely pathological gall-bladder is generally productive of a more gratifying clinical result than is excision of an organ in which the microscopic appearances are equivocal. It is desirable, without doubt, to operate before long-standing biliary disease has led to advanced secondary changes in other organs, but one will often suffer disappointment if one concludes, from mild or atypical symptoms and from doubtful pathological changes, that a case is one of early cholecystitis, suited for cholecystectomy. In any particular case, however, the responsibility of the gall-bladder for symptoms seems to be not quite proportionate to its deviation from histological normality. This is shown by the fact that a third of the cases of chronic catarrhal cholecystitis were unimproved, while a fifth of those with minimal lesions were actually relieved of all their complaints. These arguments assume that a train of symptoms originally arising from a lesion in the gall-bladder will be alleviated by its removal. This, of course, is not necessarily the case, and, on the other hand, it might well be that in some cases symptoms would actually be caused by the removal of the gall-bladder, especially if it had retained a proportion of its normal function.

The improvement in some of the cases where a minimally diseased gall-bladder was removed may have had nothing to do with the cholecystectomy, for

the symptoms may have arisen in a transient upset of some other part of the alimentary tract, and so have pursued a self-limiting course independent of the treatment exhibited. The majority, however, cannot be explained in this way. It must be remembered, in gall-bladder disease, that lesions of equal severity do not necessarily give rise to equally severe symptoms in different individuals. Something must depend upon the nervous sensitivity of the patient. Some women carry gall-stones to the grave with never an attack of colic, others have many colics, apparently without gall-stones. Possibly quite minor abnormalities in the valvular exit from the gall-bladder may dam back the fluid content and so give rise to colic, just as renal colic and hydronephrosis may result from the presence of a valve at the pelvi-ureteral junction. It is even possible that the disturbance of sensation known as biliary colic may have its origin occasionally in other sites than the gall-bladder. The same reservation may apply in even greater degree to the minor symptoms attributed to cholecystitis. These hypotheses would help to explain both the absence of histological changes in the gall-bladder in some cases with definite biliary symptoms and the occasional persistence of pain after cholecystectomy.

THE PERSISTENCE OF PAIN AFTER CHOLECYSTECTOMY

The Germans have devoted much attention to the nervous origin of biliary pain, following the lead of Westphal (1923). Principally by experiments in which he used the Rehfuess duodenal tube, as in the method of Lyon and Meltzer for non-surgical biliary drainage, this investigator arrived at the conclusion that there are three motor elements in the biliary tract, the gall-bladder musculature, the sphincter at the gall-bladder neck, and the sphincter of Oddi at the lower end of the common duct. He suggested that any upset of the co-ordinated activity of these motor elements might give rise to pain, even when the gall-bladder showed no sign of organic disease. Westphal's ideas have not been generally accepted, and although his writings are attractive and his selected case records are impressive, there is a lack of complete statistical information. As a final criticism of this work, it may be mentioned that Halpert's microscopic preparations show conclusively that in many human subjects there is no definite sphincter of Oddi. More statistical anatomical work is needed upon the musculature of the biliary tract. It would be interesting to know whether cases without a sphincter of Oddi are less subject to biliary colic. Various other suggestions have been made from time to time to account for the persistence of distressing symptoms after cholecystectomy. Some authors lay the responsibility upon secondary changes in other organs. Hepatitis, cholangitis, pancreatitis, functional and organic disturbances of stomach, duodenum, or colon, and hypercholesterolaemia have all been indicted. The majority of the cases in this paper might fairly be regarded as examples of early and slight gall-bladder disease, and so secondary changes in other organs are less likely to have been present than, for example, in old-standing cholelithiasis. Since the present series is made up of stoneless cases it is unnecessary to take up the vexed question of relict stone, new-formed stone, or recrudescing symptoms without stone.

In general, the suggestion of Dr. Evarts Graham (1933), that, in the incompletely relieved cases, the gall-bladder was not originally responsible for all of the symptoms, is the most reasonable one.

The study of the correlation of pathological changes in the gall-bladder with the ultimate condition of the patient appears to justify certain definite conclusions regarding the value to the clinician of microscopic examination of the gall-bladder in cases of cholecystitis without stone. These conclusions are as follows —

1 Histological changes in the gall-bladder are *not* an accurate index of the symptoms which have been produced by the latter. In any particular case, however, some assistance in assessing the responsibility of a stoneless gall-bladder for the symptoms may be obtained if the microscopic findings are evaluated in a general fashion, without attributing importance to slight deviations from the accepted normal histological picture.

2 Since this responsibility may not extend to all the complaints, relief following cholecystectomy may be only partial, but there is no way in which the pathologist can predict accurately the degree of improvement to be expected.

3 Undoubtedly, severe biliary symptoms may occasionally disappear after cholecystectomy despite the fact that the gall-bladder departs only slightly from normal, but certainly the more diseased the latter is, the more likely is the outcome to be happy. If cure be expected regularly whenever a minor lesion is found in the gall-bladder, disappointment will follow more often than not.

4 No support has been found for the view that the gall-bladder should be removed in the earliest stages (if such they be) of disease.

5 With regard to cholesterosis of the gall-bladder, no evidence has been obtained that this condition *per se* is liable to produce symptoms.

CHOLECYSTOGRAPHY IN THE PRESENT SERIES

The Relation between Variations in Cholecystographic Findings and Pathological Changes in the Gall-bladder—The radiological test known as cholecystography rests upon the work of Graham and Cole (1924). From the two premises (1) that certain phthaleins are excreted in the bile, as shown by Rowntree and his co-workers, and (2) that the gall-bladder concentrates the bile, these investigators developed the argument that if the phthalein could be made radio-opaque by halogenation, then the gall-bladder might be visualized.

The physiological basis of cholecystography is the subject of dispute, but it is clear that, whatever the basis, cholecystography enables us to visualize a living process in which the mucous membrane of the gall-bladder is intimately concerned, and it will undoubtedly prove valuable if we can convert the information obtained into terms of pathological changes in the mucosa.

Table VII represents an analysis showing the various types of cholecystographic response encountered in the various categories of pathological lesions of the gall-bladder. It should be understood clearly that in those cases in which the cholecystogram is termed 'deformed' the density of the dye shadow was normal, but the outline of the gall-bladder was irregular, a state of affairs attributed to pericholecystic adhesions.

The figures and the terms are almost self-explanatory. The general result is that the great majority of the cases fall into the category "gall-bladder faintly visualized." The figures in the series of cases of simple cholesterosis do not differ markedly from those in cases histologically almost normal. This fact is further evidence against the existence of a definite inflammatory basis for lipoid infiltration of the gall-bladder. In the presence of marked lesions—namely, fibrous

cholecystitis or stones—no case concentrated the dye satisfactorily, and those of chronic catarrhal cholecystitis showed a tendency to cholecystographic impairment intermediate in degree. It is clear, therefore, that there is no type of cholecystographic reaction quite characteristic of any particular type of pathological change, although, as one would expect, the more advanced the pathology, the less is the chance of the dye being concentrated normally.

Table VII—THE CORRELATION OF THE CHOLECYSTOGRAM WITH PATHOLOGICAL CHANGES IN THE GALL-BLADDER, IN 181 STONELESS CASES AND 20 CASES OF CHOLESTEROSIS WITH BILIARY CALCULI

PATHOLOGY	TOTAL	CHOLECYSTOGRAM				PERCENTAGE IN WHICH GALL-BLADDER SHOWED NORMAL CONCENTRATION OF DYE (i.e., 'normal' and 'deformed')
		Normal	Deformed	Faint	No Shadow	
Minimal lesion	76	9	11	49	7	26
Cholesterosis	38	6	3	25	4	25
Chronic catarrhal cholecystitis	58	6	2	36	14	14
Chronic fibrous cholecystitis	9	0	0	3	6	0
Total of stoneless cases	181	21	16	113	31	20
Cholesterosis with stone	20	0	0	17	3	0

Provided that cholecystography is to be regarded as a test of the function of the gall-bladder, these results may be used further to support an attitude of scepticism regarding the importance of minor histological lesions, for if a gall-bladder which is so abnormal as to be classed as 'chronic catarrhal cholecystitis' may give on occasion a normal concentration of the dye, the significance of a few lymphocytes in cases classed as 'minimal lesion' must surely be debatable. Conversely, if we are to attribute significance to microscopic appearances, then the present findings fail to support statements made in the early period of cholecystography, that absence or faintness of the shadow invariably indicates a pathological gall-bladder.

It is necessary to seek an explanation of the fact that in seven of the cases in which the gall-bladder lesion was minimal (see, for example, Fig 187), no shadow was obtained by cholecystography, especially since the cholecystogram was an important element in the evidence leading to cholecystectomy.



FIG 187—Unexpectedly normal histological findings in a case in which no shadow was obtained by cholecystography. The gall-bladder and liver both seem practically normal. The marked lysis of the lining epithelium is of course a post-operative phenomenon. ($\times 50$)

From the time of its introduction, it has been emphasized that the Graham-Cole test demands an adequately functioning liver. In twenty-eight consecutive examples of enlarged liver due to various causes, Alexander and Bond, of St Louis, were unable to visualize the gall-bladder (Moore, 1933). Moore states that, "Whether or not intensive disease of the gall-bladder is essential for the ablation of its concentrating function is not known, and on the other hand it is unknown whether loss of concentrating function is precedent or contributory to a chain of pathological changes which might eventuate in infection or stone formation." Again, "It is possible that disease of the appendix or other remote part of the alimentary canal might conceivably affect the gall-bladder to the point of destroying the concentrating function, as such lesions unquestionably affect alimentary secretions."

Another point that should be emphasized as a possible explanation of occasional non-visualization of relatively normal gall-bladders is the well-known fact that such failure of visualization may be quite temporary. Accordingly, unless the history points fairly definitely to gall-bladder disease, failure to obtain a shadow should not be accepted as conclusive evidence against the gall-bladder until the test has been repeated with confirmatory result. I am not aware that this is at all standard practice except in so far as a doubtful result after oral administration of the dye is sometimes checked by an intravenous test.

With regard to the 'faintly visualized' gall-bladders, it may be mentioned that Kirklin (1933) does not regard these as pathological provided the gall-bladder shadow is denser than that of the liver.

The conclusion to be drawn from this section of the study is that in cases of cholecystitis without stone, the cholecystogram corresponds only roughly with the histological findings in the gall-bladder, and occasionally the two sets of findings are difficult to reconcile. This does not imply that more importance should be attached to either one or other of the methods of investigation.

The Cholecystogram as an Index of Prognosis in the Stoneless Gall-bladder Treated by Cholecystectomy.—In many cases of the present series in which the clinical features were vague or atypical, the cholecystogram was an important factor in establishing the decision to remove the gall-bladder, and the correctness of these decisions may be checked by an analysis of the relation between the type of the cholecystographic response and the clinical result of cholecystectomy (*Table VIII*).

Table VIII—THE RELATION BETWEEN CHOLECYSTOGRAM AND END-RESULT AFTER CHOLECYSTECTOMY IN 116 CASES, ALL STONELESS

TYPE OF CHOLECYSTOGRAPHIC RESPONSE	TOTAL	CLINICAL RESULT				
		Well	Improved	Unimproved	Post operative death	Percentage well or improved
Normal	10	5	1	4	0	60
Deformed shadow	8	4	2	2	0	75
Faint shadow	76	22	23	28	3	59
No shadow	22	6	7	6	3	60
TOTALS	116	37	33	40	6	60

The figures show that cure or improvement followed operation in 60 per cent of all the cases. The smaller groups are really inadequate for detailed analysis, but it is seen that the probability of benefit from the operation was not greater in the cases in which no shadow was obtained than in those in which, according to this test, the gall-bladder was normal. This is a surprising and disappointing result, and its negative nature must be partially explicable as coincidence, due to the small number of 'normal cholecystogram' and 'no shadow' cases. The only group which stands out is that in which the shadow, though of satisfactory density, was deformed, of eight such cases, six were cured or partially relieved of their complaints as a result of operation. Deformity of the gall-bladder shadow is generally associated with dense pericholecystic adhesions, and the excellence of the results obtained in cases of this class, as compared with others in which pericholecystitis is less marked, has already been pointed out.

It is clear that much remains to be learned regarding the gall-bladder in health and disease. Cholecystography is a test of one of the functions of the gall-bladder, but the physiological factors which determine a normal reaction are not certainly established, and the factors which lead to a departure from the normal response are even less clear, and may well be manifold. Accordingly, while experience has proved that advanced disease of the gall-bladder will impair or prevent the development of the gall-bladder shadow, there is not yet reason to be sure that the converse is equally true. Diminution of intensity of the gall-bladder shadow need not necessarily imply that the organ is pathological, nor that it is responsible for the abdominal symptoms complained of by the patient, unless these are quite typically of biliary type. Much more work remains to be done before disease of the gall-bladder can be recognized cholecystographically with precision.

Impairment of the density of the cholecystographic shadow is not of itself evidence that removal of the gall-bladder will be followed by a satisfactory clinical result

GENERAL CONCLUSION

From a detailed study of 243 cases of cholecystitis without stone and 21 cases of cholesterosis of the gall-bladder accompanied by gall-stones, the following conclusions have been reached —

In cases of cholecystitis without stone, cholecystectomy carries a mortality of 3 per cent. Cure of symptoms results in 30 per cent, and improvement in 30 per cent. In 37 per cent the end-result of operation is unsatisfactory.

Cholecystitis without stone seems to belong to a region on the borderline between functional and organic disease.

No single test is infallible, though in the individual case study of the clinical history, of the cholecystogram, and subsequently of the microscopic sections may each yield information pointing toward or away from the gall-bladder.

The results of cholecystectomy indicate that it is dangerous to over-emphasize any one of these methods of investigation, or to attribute importance to minutiae.

To establish a diagnosis of cholecystitis the history must be typical, and should include pain. No evidence has been obtained that either flatulent dyspepsia or 'food selection' necessarily indicates gall-bladder disease or is likely to be relieved by removal of the gall-bladder.

Cholecystographic changes must be definite, and mere impairment of the

density of the gall-bladder shadow does not invariably mean that the gall-bladder is pathological. While, as is well known, almost every gall-stone will be revealed by the use of the dye test, the same degree of precision is not attained in the field of stoneless cholecystitis.

Microscopic changes are probably not significant unless they are fairly gross. The rather surprising conclusion has been reached that cholesterosis of the gall-bladder is not of itself a pathological or symptom-producing condition.

A considerable proportion of patients are unrelieved by removal of a stoneless gall-bladder, the therapeutic failures are due to symptoms having their origin outwith the gall-bladder. The appendix, however, does not seem frequently to be the organ responsible.

The results of surgical treatment of cholecystitis without stone are relatively unpredictable in the individual case, even by the most modern of laboratory procedures. Certainly they are not so good as in the presence of gross organic disease, when, generally speaking, symptoms are clamant and relief after operation is dramatic.

This work was done during 1932-3, while the author held a Rockefeller Foundation Travelling Fellowship in Surgery, and in the first place the warmest thanks are due to the Rockefeller Foundation for Medical Research. Material for the study was obtained at Washington University Medical School, St. Louis, Mo., and the investigation was facilitated by the wholehearted co-operation of the staff of Barnes Hospital. Records were made available without restriction, and help given in the tracing of cases. Dr. Evarts A. Graham gave in addition much time and interest to the planning of the investigation and to its direction while in train. Dr. Sherwood Moore, of the Edward Mallinckrodt Institute of Radiology, was generous of his time in the discussion of radiological interpretation. Miss Gertrude Cassell, of the Department of Surgical Pathology, prepared numerous excellent histological specimens. Miss Dorothy Blanchard and Miss Ada Hanvey gave unstinted assistance in the tedious secretarial work involved in the follow-up study, and Miss Agnes Kelly, of Glasgow, in the final compilation.

REFERENCES

- ALVAREZ, W. C., MEYER, K. F., RUSA, G. Y., TAYLOR, F. B., and EASTON, J., "Present-day Problems in Regard to Gall-bladder Infections", *Jour Amer Med Assoc*, 1923, lxxxi, 974.
- CARMAN, R. D., MACCARTY, W. C., and CAMP, J. D., "Roentgenologic Diagnosis of Cholecystic Disease", *Radiology*, 1924, ii, 80.
- CONNELL, F. G., "Remote Results of Biliary Surgery", *Ann of Surg*, 1928, lxxviii, 837.
- DEAVER, J. B., "Gall-bladder Disease", *Surg Clin N Amer*, 1925, v, 1516.
- DEAVER, J. B., and BORTZ, E. L., "Gall-bladder Disease, a Review of 903 Cases", *Jour Amer Med Assoc*, 1927, lxxxviii, 619.
- FEINBLATT, H. M., "The Infrequency of Primary Infection of the Gall-bladder", *Trans Amer Gastro-enterol Assoc*, 1928, 203.
- FLORCKEN, H., "Die 'Ruckfalle' nach Gallensteinoperationen", *Munch med Woch*, 1923, lxx, 498.
- GRAHAM, E. A., "Lowering the Mortality after Operations on the Biliary Tract", *Illinois Med Jour*, 1931, Sept.
- GRAHAM, E. A., Personal communication, 1933.
- GRAHAM, E. A., and COLE, W. H., "Roentgenologic Examination of the Gall-bladder. Preliminary Report of a New Method utilizing the Intravenous Injection of Tetra-bromphenolphthalein", *Jour Amer Med Assoc*, 1924, lxxii, 613.

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- GRAHAM, E A, COLE, W H, COPER, G H, and MOORF, S, "Simultaneous Cholecystography and Tests of Hepatic and Renal Function", *Ibid*, 1926, lxxxi, 467
- GUNDERMANN, W, "Beitrag zur Bakteriologie und Pathologie der chirurgischen Erkrankungen der Gallenwege", *Mitt aus d Grenzgeb Med u Chir*, 1924, xxxvii, 243
- HARTMANN, H, and PETIT-DUTAILLIS, D, "Les Suites eloignees de la Cholecystectomie", *Jour de Chir*, 1922, xx, 349
- HIRSCH, I S, and TAYLOR, H K, "Gall and Gall-bladder Diagnosis", *Radiology*, 1928, xi, 37
- HITZROT, J H, and CORNELL, N W, "An Analysis of 482 Gall-bladder Cases", *Ann of Surg*, 1926, lxxxiv, 829
- HOTZ, G, "Ergebnisse der Gallensteinchirurgie", *Arch f klin Chir*, 1923, cxxvi, 284
- HUECK, H, "Zur Frage der Kolkrezidive nach Cholecystektomie", *Arch f klin Chir*, 1927, clxvi, 255
- JUDD, E S, "Clinical versus Pathological Cholecystitis", *Collected Papers of the Mayo Clinic*, 1925, xvii, 152
- JUDD, E S, "Cholecystitis", *Ibid*, 1927, xix, 324
- JUDD, E S, and BURDEN, V G, "Non-calculous Biliary Obstruction following Cholecystectomy", *Ann of Surg*, 1924, lxxix, 533
- JUDD, E S, and PRIESTLEY, J T, "Ultimate Results from Operations on the Biliary Tract", *Jour Amer Med Assoc*, 1932, xcix, 887
- KEHR, H, "Die Praxis der Gallenwege-Chirurgie", 1913, i, 315 Munich J F Lehmann
- KIRKLIN, B R, Personal communication, 1933
- LAHEY, F H, "Cholecystitis, the Cholesterol Gall-bladder, and Silent Gall-stones", *Boston Med and Surg Jour*, 1927, cxcvi, 677
- MOORE, SHERWOOD, Personal communication, 1933
- MOYNIHAN, B G A, "A Disease of the Gall-bladder requiring Cholecystectomy", *Ann of Surg*, 1909, i, 1265
- MULLER, G P, "The Non-calculous Gall-bladder", *Jour Amer Med Assoc*, 1927, lxxxix, 786
- ROSS, J C, "The Symptomatic End-results of Operations for Cholecystitis", *Brit Med Jour*, 1932, i, 1026
- SCHMIEDEN, V, and ROHDE, C, "Die Stauungsgallenblase mit besonderer Berücksichtigung der Aetologie der Gallenstauung", *Arch f klin Chir*, 1921, cxviii, 14
- SCHONDUBE, W, "Ueber Dysfunktionen stein- und entzündungs-freier Gallenblasen", *Zeits f klin Med*, 1928, cix, 447
- SEULEBERGER, P, "Nachuntersuchungen der in der Göttingener Klinik operierten Gallensteinkranken", *Deut Zeits f Chir*, 1925, cxc, i
- STANTON, E MACD, "The Stoneless Gall-bladder A Study of Operative Cases", *Amer Jour Surg*, 1932, xlviii (NS), 246
- WESTPHAL, K, "Muskelfunktion, Nervensystem und Pathologie der Gallenwege", *Zeits f klin Med*, 1923, xcvi, 22
- YOUNG, E L, Jun, "The End-results of Chronic Cholecystitis", *New Eng Jour Med*, 1928, cxviii, 729
- ZINK, O C, "A Clinical Study of Cholecystitis with the Aid of Cholecystography", *Radiology*, 1926, vi, 286

PANCREATIC FISTULA

REPORT OF A CASE CURE BY PANCREATO-GASTROSTOMY*

By ROBERT M. JANES, TORONTO

PANCREATIC fistulæ result from injuries to the pancreas, from the treatment of cysts of the pancreas by marsupialization, or from extensive necrosis of the gland. Few of these fistulæ persist. Of thirty-three pancreatic cysts treated by marsupialization by Judd, Mattson, and Mahorner,¹ none required further treatment. In some the discharge continued for a few weeks only, in others for a few months, and in a few for a year. The maximum period of discharge was two years. Injuries are likely to produce a niche in the gland where it is squeezed against the anterior surface of the vertebra, and not an injury to the main duct. It seems probable that, as suggested by Kahn and Klein,¹³ spontaneous closure is likely to occur unless the continuity of the duct of Santorini is interrupted, in which case the secretion from the portion of the pancreas distal to the point of injury can escape only by way of the fistula.

Only in those cases in which the communication with the pancreas is so located that the greater portion of the secretion escapes by way of the fistula is digestion likely to be seriously interfered with. In the case to be reported not more than one-fourth of the gland retained its connection with the intestine, yet at no time was there evidence of failure of pancreatic digestion. It has been shown experimentally that loss of the total supply of pancreatic juice is rapidly fatal.^{11 12} When the loss of secretion is great the collected discharge should be fed to the patient. When practically moribund, experimental animals may be restored to an apparently normal state by the giving of whole pancreatic juice by the mouth.¹¹ Cathala and Seneque³ demonstrated an equally beneficial effect in man. The lives of animals in which complete fistulæ have been established can be prolonged greatly by the administration of large quantities of normal saline. In the present case the administration of normal saline seemed sufficient to maintain the general health owing presumably to the fact that enough secretion continued to be discharged into the intestine.

Since pancreatic juice may be activated by contact with any tissue, a rapid digestion of the abdominal wall is likely. This may be controlled by the removal of the secretions from the fistula by way of a catheter to which a suction pump is attached or by inactivation of the secretion as suggested by Potter,^{6, 7} through the continuous addition to it of sufficient N/10 HCl to render its reaction acid and the use of a dressing of 10 per cent Witte's peptone to take up any enzymes not rendered innocuous in the acid medium. Removal of the secretion by suction proved entirely satisfactory in this case. As much as 20 oz. was collected daily, all three enzymes

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were present and active. The skin about the mouth of the fistula was protected by a paste of bismuth in olive oil.

Since spontaneous closure of a pancreatic fistula may occur up to one year and even occasionally two years, it would seem that at least the former period should be allowed to elapse before any radical surgical measures are undertaken. Serious impairment of health as a result of the loss of pancreatic juice, and the certainty that the fistula constitutes the only possible escape for secretion from the portion of gland distal to it, would appear to be exceptions to this rule.

Two possible methods of surgical attack suggest themselves: first, excision of the fistula along with the portion of pancreas discharging into it, and, second, implantation of the mouth of the fistula into an adjacent hollow viscus. The former is much too dangerous and has been rejected in favour of the implantation, which is comparatively safe. Implantation into the stomach, gall-bladder, duodenum, and jejunum has been done, but so far as one can discover it has been done successfully only into the stomach. Implantation into the stomach would appear to be preferable because of the ease with which it may be carried out and because the secretion when emptied into an acid medium is at once inactivated. There does not seem to be any convincing evidence that radium treatment is of value. Corachan,⁴ writing in 1928, was able to collect six cases in which implantation into the stomach had been done successfully, one each by Doyen (1905), Michon (1911), Jedlik (1921), Gutierrez (1925), Courboulis (1926), and his own in 1927. Cathala and Seneque³ reported a seventh successful case in 1930. The following appears to be the eighth successful case to be reported.

HISTORY—Mrs E M, aged 54 years, was admitted to the hospital on May 20, 1932. Four years previously she had had an attack of severe upper abdominal pain which confined her to bed for three weeks. The stools were light-coloured and the urine dark, but she had not been jaundiced. During the following years she had remained well apart from some indigestion. Two weeks before admission she was wakened at night with terrific pain in the epigastrium, frequent vomiting occurred. A physician was called who administered hypodermics. During the next three days a continuous pain remained, with periods of increased severity. The physician called twice daily to relieve the pain by hypodermics. She continued to vomit dark brown material. During the following ten days the pain persisted but was less severe, she developed a fever.

ON ADMISSION—When admitted to hospital the patient was fairly comfortable. The temperature was 103°, the pulse 100, blood-pressure 104/62, and the white blood-cells 21,000. The specific gravity of the urine was 1013, it was acid, and contained no albumin and no sugar.

ON EXAMINATION—The abdomen was moderately rounded. There was some general rigidity of the upper abdomen with tenderness in the right hypochondrium, where an indefinite mass which was regarded as a distended gall-bladder could be felt. A diagnosis of cholecystitis and cholelithiasis with possible pancreatitis was made. During the following week there appeared to be some improvement.

On May 29 at 7 p.m. she experienced severe epigastric pain and vomited about four ounces of coffee-ground material. Examination at this time revealed a rounded, rather sharply delimited mass about the size of a grape-fruit which filled

the whole epigastrium The vomiting of coffee-ground material continued, and by 8 p.m. the blood-pressure was 80/35 and a little later 70/35, the pulse-rate increased to 150 per minute Her condition slowly improved, and when seen by me at 5 p.m. the following day it was not alarming The previously described mass was easily palpated and its position between the liver and stomach, pushing the latter downward and to the left, was confirmed by a small barium meal (*Fig 188*) The diagnosis of acute pancreatic necrosis with hæmorrhage seemed obvious

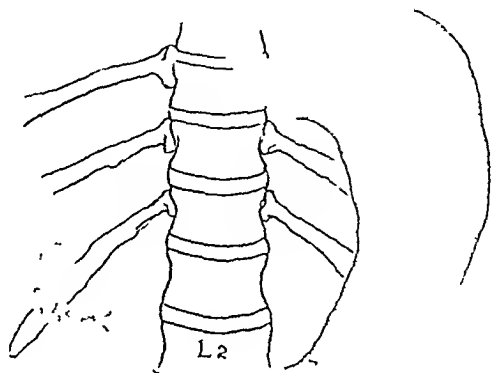


FIG 188—Stomach displaced downward and to the left by mass rising above the lesser curvature, and filling defect in the stomach produced by the enlarged pancreas

FIRST OPERATION—The abdomen was opened at 8 p.m. under spinal anaesthesia There was extensive fat necrosis, a mass the size of a grape-fruit bulged forward from behind the lesser omentum The head and tail of the pancreas were both greatly enlarged and stony hard The gall-bladder contained three rather large stones, the cystic duct contained no stone, the common duct was small and contained no stone The lesser omentum and posterior peritoneum were incised and about 10 oz of blood-clot were evacuated, then a greyish mass of pancreas 4 by 1½ by 1 in It was now evident that the middle third of the pancreas had been removed as a slough A ½-in tube containing a gauze wick was introduced into the lesser sac, the gall-bladder emptied and drained, and the abdomen closed

The following day the patient was remarkably well There was a free discharge of bile from the gall-bladder, and opalescent fluid in large quantities, at first blood-stained, escaped from the lesser sac On June 4, because the skin of the abdomen was becoming excoriated, a suction pump was attached

Drainage from the pancreatic fistula reached a maximum of 20 oz daily It contained no bile until the gall-bladder drain was removed on June 22, following which the discharge was bile-stained The fluid collected gradually decreased in amount up to July 22, at which time all tubes were removed and the suction was discontinued The patient was allowed home on Aug 11 A fistula the size of a 10 F catheter persisted from which a little limpid fluid escaped

RE-ADMISSION—On Sept 11 she was re-admitted with the history that on three occasions since discharge the drainage had ceased and she had coincidentally developed pain in the epigastrium and vomiting, relief was obtained by re-opening the fistula. During the next two months this occurrence was repeated many times. By Dec 12 a mass the size of an orange had formed in the epigastrium. She was allowed home again. On Jan 16, 1933, she was re-admitted with a history of having been fairly well since last seen. The fistula had closed, but the mass in the epigastrium was now two-thirds the size of an association football (*Fig 189*). A little bile-stained mucus was escaping from the gall-bladder.

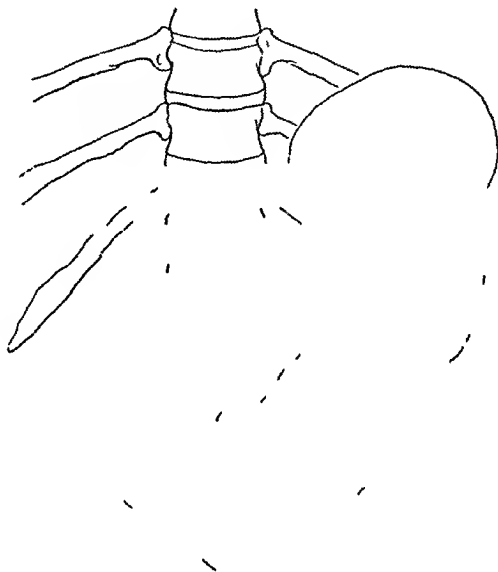


FIG 189—Displacement of stomach produced by pancreatic cyst

SECOND OPERATION—On Jan 27, under spinal anaesthesia, the pancreatic cyst was exposed, opened, and 1200 c.c. of slightly yellowish fluid were evacuated from it. The cyst wall was thin and bluish in colour. Two ounces of pure formalin were introduced and allowed to remain in contact with the lining of the cyst for five minutes. The cyst was marsupialized.

During the next few weeks there was a discharge of grumous material with sloughs of cyst lining. The fluid became increasingly clear, and by March 16 12 oz. of clear fluid were being discharged daily, which, despite the fact that it was active in pancreatic ferments, showed no tendency to digest the skin. This was due, presumably, to the fact that it was escaping along a dense fibrous channel which did not activate the enzymes. On April 12 lipiodol was injected into the fistula and a clear outline of the tail of the pancreas obtained (*Fig 190*). There was transient abdominal pain following this.

THIRD OPERATION—On April 18 the patient was again operated upon under spinal anaesthesia. It proved easy to dissect the fibrous fistulous tract as far as its origin from the pancreas. It was implanted into an adjacent area on the posterior wall of the stomach. In order to rid the patient of a small biliary fistula

the gall-bladder was removed. A $\frac{1}{4}$ -in rubber drainage tube was placed above the stomach to the site of implantation and the abdomen closed.

The post-operative course was uneventful. The drain was removed at the end of forty-eight hours and the wound healed by primary union. The patient was discharged from hospital at the end of three weeks and has remained well. She has no indigestion and no abdominal discomfort. Barium examination of the stomach now demonstrates no deformity or other evidence of the presence of the pancreato-gastrostomy (Fig 191).

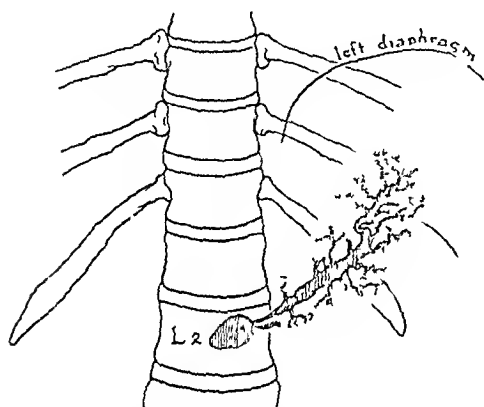


FIG 190—Outline of pancreas obtained by injecting lipiodol into the fistula

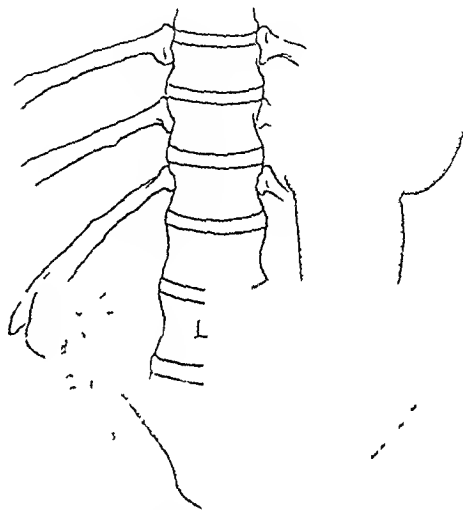


FIG 191—Barium meal demonstrates no deformity of the stomach or other evidence of the presence of the pancreato-gastrostomy

BIBLIOGRAPHY

- ¹ JUDD, E S, MATTSON, H, and MAHORN, H R, *Arch of Surg*, 1931, **lxii**, 838
- ² ISNARDI, N, and ZANARDI, J, *Semana med*, 1925, Nov (Abstr *Jour Amer Med Assoc*, **lxxxvi**, No 11, 809)
- ³ CATHALA, J, and SENEQUE, J, *Presse med*, 1930, Nov 12, 1534
- ⁴ CORACHAN, M, *Ibid*, 1928, **xxxvi**, 1394
- ⁵ BICKHAM, *Operative Surgery*, 1924, **iv**
- ⁶ FAST, W K, *Jour Amer Med Assoc*, 1930, Nov 29, 1668
- ⁷ POTTER, C, *Jour of Missouri State Med Assoc*, 1932, **xxix**, 374
- ⁸ HAMILTON, C S, *Surg Gynecol and Obst*, 1922, **xxxv**, 655
- ⁹ CULLER, R M, *Jour Amer Med Assoc*, 1920, **lxxv**, July 3
- ¹⁰ MEYER, F, *Med Jour of Australia*, 1916, **iii**, 396
- ¹¹ McCAUGHAN, J M, *Proc Staff Meetings of Mayo Clinic*, 1929-30, **iv-v**, 199
- ¹² DRAGSTEDT, L R, MONTGOMERY, M L, and ELLIS, J C, *Proc Soc for Exper Biol and Med* 1930, **xxviii**, 109
- ¹³ KAHN, J, and KLEIN, H M, *Amer Jour Med Sci*, 1932, **clxxxiv**, 503

TORSION OF THE GALL-BLADDER

BY A RENDLE SHORT

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ALTHOUGH torsion of the gall-bladder is reputed to be a rare condition, it ought not to be left out of mind by the surgeon. We have had three cases under our care within the past four years, so it seems probable, as with other so-called 'rare' conditions, that a good many cases go unreported. Four surgeons have reported two each, and one (Henschen, of St Gall) has seen three.

We believe that it should often be possible to make a diagnosis before operation, and if this is done it will probably open the way to save the patient's life by timely surgery.

Although nine cases have been described by British surgeons in addition to our own, there does not appear to be any detailed account of this disease in English literature, and widely read text-books of surgery which we have consulted make little or no reference to it.

HISTORY

The first case on record was published by A V Wendel in America in 1898, and is, with one exception, the youngest patient reported, also it is the only one in which the gall-bladder perforated. Ten years later, three cases were described from Germany. The best descriptions of acute torsion of the gall-bladder are those of A M Shipley (American), P Costantini (Italian), and P Brocq (French). A table of the recorded cases is given on pp 302-4.

Cases have also been described by the following observers, but we have not been able to consult the accounts, as the literature was not accessible to us —

Henschen, 1915 (three cases)
Daux, 1924 (girl of 11)
Lutaud, 1924
Ceccarelli, 1926 (two cases)

Daraignez, 1926
Ramshorst, 1926
Romani, 1928 (this patient had appendicitis as well)
Meeker and Liserby, 1932

In 1925 A Fischer, of Budapest, described two cases of recurrent and incomplete torsion of the gall-bladder, both in women, aged 44 and 29 respectively. Both complained of repeated attacks of pain in the right hypochondrium, without jaundice, and both suffered from general visceroptosis. They were operated on in a quiescent interval and did well. In both cases stones were present, and the torsion was through 90°. In one case the gall-bladder was removed, and in the other it was anchored by sutures. Fischer quotes another case reported by Seefisch, who also operated and anchored the gall-bladder by suturing.

ACUTE CASES OF TORSION OF THE GALL-BLADDER

AUTHOR	YEAR AND PLACE	AGE	SEX	DESCRIPTION	STOVES	TREATMENT	RESULT
Wendel, A V	1898	23	F	Lump found in November, 1895 Attack of pain in 1896 Operation in 1897	213	G B twisted and perforated G B removed	Recovered
Mayer, W	1908, Mannheim	54	F	5th day Acute pain Tumour Pulse 150 Diagnosis intestinal obstruction, due to hydronephrosis	Yes	G B removed	Died
Muhsam, R	1908, Berlin	61	F	Pulse 96, temp 99° Tumour felt under ether, not before Diagnosis appendicitis	No	G B removed	Recovered
Nehrkorn	1908	74	F	Long history Operation on 8th day Diagnosis empyema of gall-bladder	No	G B removed	Recovered
Lett, H	1909, London	72	F	3rd day Temp 101°, pulse 100 Tumour felt Diagnosis cholecystitis	Yes	Mesentery present G B removed	Died
Fischer, A	1910, Darmstadt	70	F	3rd day Tumour felt Diagnosis cholecystitis or appendicitis	No	G B removed	
Kubig, G	1912	73	M	Died of phthisis	No	P M case	
Cramp, W C	1915, New York		M	Temp 99° Rigidity and pain lump	No	G B removed	Recovered
Reichel	1919, Chemnitz	71	F	3rd day Pulse 88 Pain, vomiting No lump Diagnosis appendix	No	Twist clock-wise removed	Recovered
Krabbel, Max	1920	73	F	4th day Temp 98° Tumour Diagnosis intestinal obstruction from cancer of colon Previous attacks of gall-stone colic hours' history Distended Tumour Pain and vomiting Diagnosed correctly before operation	One	G B removed G B removed	Recovered Recovered
Strauss, M	1921	42	F	Pain, vomiting, constipation Tumour Temp 98°, pulse 108 Diagnosis cholecystitis + hydrops	One	G B removed	Recovered
Hansen, P N	1921	79	F	24 hours Tumour			

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TORSION OF THE GALL-BLADDER									
Author	Year	Age	Sex	Duration of Illness	Chief Complaints	Physical Examination	Diagnosis	Treatment	Result
Frankau, C H S	1922, London	62	F	24 hours	Pain ing, constipation	Temp 97.6°, pulse 112	No tumour	Vomiting	Recovered
Irwir, S I	1922, Belfast	34	F	26 hours	Pain and vomiting	Temp 99°, pulse 90	Tender and rigid	No	Recovered
Jonas, H C	1923, Barnstaple	67	F	24 hours	Pain, vomiting	Temp 99°, pulse 94	Tumour	Yes	Recovered
Dubs, J	1924, London	54	F	48 hours	Pain, pulse 94	Ill several days with normal, a few days	Normal	No	Recovered
Fifield, L R	1925, London	71	F	Previous attacks	Pain, pulse 108	Normal, a few days	Normal	No	Recovered
Huddy, G P B	1926, Birmingham	71	F	Previous attacks	Pain, pulse 108	Normal, a few days	Normal	No	Recovered
Grunert, A	1926, Berlin	66	F	2 days	Pain, vomiting	Temp 99°, pulse 110	Normal	No	Recovered
Sutter, A	1925	74	F	Previous attacks	Sudden pain, vomiting	Temp 99°, pulse 110	Normal	No	Recovered
Marinacci, S	1926	—	F	24 hours	Pain, vomiting	Temp 99°, pulse 110	Normal	No	Recovered
Holden, W B	1927, Portland, Oregon	49	M	24 hours	Pain, vomiting	Temp 99°, pulse 110	Normal	No	Recovered
Zum Busch, J P	1927, Liestal	65	F	24 hours	Pain, vomiting	Temp 99°, pulse 110	Normal	No	Recovered
Shipley, A M	1927, Baltimore	48	F	24 hours	Pain, vomiting	Temp 99°, pulse 110	Normal	No	Recovered
Esau	1927	48	F	24 hours	Pain, vomiting	Temp 99°, pulse 110	Normal	No	Recovered
Mcade, H	1929, Dublin	36	F	24 hours	Pain, vomiting	Temp 99°, pulse 110	Normal	No	Recovered

ACUTE CASES OF TORSION OF THE GALL-BLADDER—continued

AUTHOR	YEAR AND PLACE	AGE	SEX	DESCRIPTION	STONES	TREATMENT	RESULT
Gwyne, R G	1930, Redhill	77	F	Pain, vomiting, tumour temp 98°	One	Clockwise Mesentery G B removed	Recovered
Costantini	1930, Gallarate, Lombardy	42	M	3rd day Pain, vomiting No tumour Diagnosis intestinal obstruction	No	Mesentery G B removed	Recovered
Kettner	1930, Karlsruhe	55	F	Tumour felt Temp normal Diagnosis volvulus of G B	?	Twisted 180° G B re-moved	?
Brocq, P	1931	70	F	Pain, vomiting, tumour felt normal after 36 hours Diagnosis GB Pulse and temp rising Tumour no longer felt	No	GB brought out, tube tied in	Recovered
Kowalenski	1931, Voronezh	67	F	10 days' pain Pulse 90, temp normal Tumour Diagnosis appendicitis	No	Thrice twisted Mesentery G B removed	Recovered
Rouhier	1931	78	F	Diagnosis intestinal obstruction	?	Anti-clockwise G B re-moved	Recovered
Leriche	1931	—	—	Diagnosis acute cholecystitis	?	Bilobular Fundus twisted G B removed	Recovered
Herfarth, H	1932, Glogau	46	F	Pain 6 weeks Temp normal Pain, tenderness, no tumour	No	Distal part of G B twisted G B removed	Recovered
Murray, J F	1932, Dundee	83	M	3rd day Pain, vomiting, rigidity, tenderness, no tumour	No	Clockwise G B re-moved, easy	Died 6 weeks later
Rendle Short	1929, Bristol	69	M	4th day Pain, rigidity, tenderness No tumour felt till under anæsthetic	No	Anti-clockwise G B re-moved	Recovered
	1933, Bristol	81	F	Pain, vomiting 1st day Temp and pulse normal Tumour which came and went Diagnosis acute G B	One	180° anti-clockwise G B removed	Recovered
Paul, R G	1933, Bristol	82	F	Pain, vomiting 1st day Temp 100°, pulse 120 No tumour Rigid, tender, Diagnosis appendicitis	One	180° anti-clockwise G B removed	Recovered

In addition to the above, two cases were described by H Krukenberg in 1903, in which kinking of the gall-bladder gave rise to attacks of colic, it was found to be long and acutely flexed on itself. It was fixed in better position, and both patients, young women, were cured. We have not reckoned these as torsion cases.

CASE REPORTS

Case 1 (A R S)—Mr W, aged 69, seen at the Bristol Royal Infirmary on June 17, 1929, complaining of pains in the chest. Four days previously the patient got up to go to work and had a pain, but managed to carry on. The next day he went to Dr Morgan, who sent him up to the Out-patient Department, and he was admitted. Pains were continuous, lasting through the night, and prevented him sleeping. No vomiting. The upper abdomen was rigid and tender. Pulse and temperature were normal. The clinical diagnosis was carcinoma of the stomach, or leaking gastric ulcer. Palpation of the abdomen under an anæsthetic revealed a lump in the liver region, which was thought to be a secondary growth.

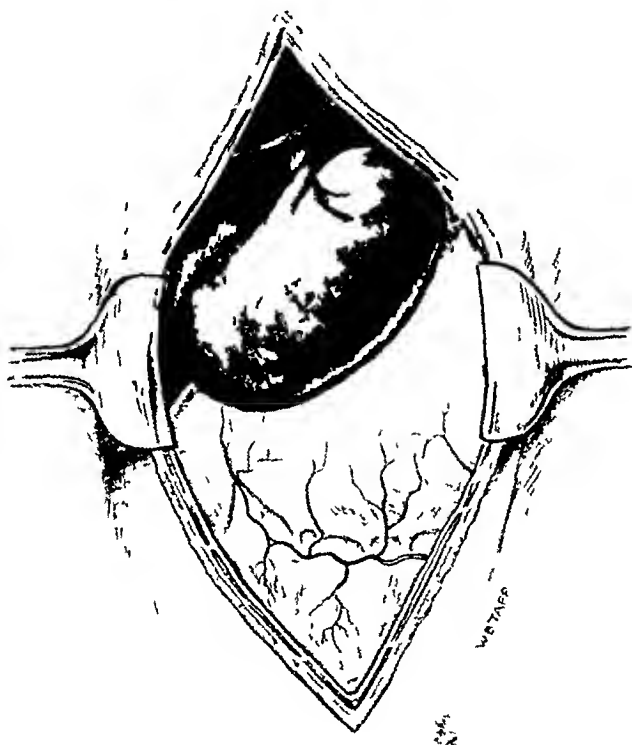


FIG 192.—*Case 1* The condition of the gall-bladder found at operation

in the liver. A small incision was made, and as it did not feel like a growth, the incision was prolonged, and revealed a large black lump attached to the liver which was found to be the gall-bladder. It was twisted on its pedicle, and was removed quite easily. Further exploration of the abdomen revealed a right hydronephrosis due to stone, which was left alone, as the patient was not in a good condition. The abdomen was closed through and through with silkworm gut. The gall-bladder was black, the wall greatly thickened, but not quite gangrenous. There were no stones. The rotation was anti-clockwise. The patient has been very well since (March, 1934). The condition is shown in Fig 192.

Case 2 (A R S)—Mrs M, aged 81, seen in consultation with Dr Fells on Oct 12, 1933. The patient had had jaundice some weeks previously, followed by pain. She was seized at 9 a.m. on the day of examination with violent colicky pain in the centre of the abdomen, some diarrhoea, and persistent vomiting. Pulse and temperature were not affected. The face was sallow, but there was no true jaundice. On examination of the abdomen a swelling about 4 in. long, having the general characters of an enlarged gall-bladder, was felt projecting below the ribs in the right hypochondrium downwards and slightly inwards. It was freely movable. The abdomen was not distended. There was slight rigidity and tenderness over the tumour. Rectal examination was negative. A tentative diagnosis was made of probable empyema of the gall-bladder, or possibly intussusception, and an immediate operation was advised.

By the time the patient had been removed to a nursing home the pain had gone, and the swelling had practically disappeared. She wanted to be let off the operation, but we advised her that it would be better to go on with it. Under the anæsthetic *no tumour could be felt*, and once again the question arose of abandoning the operation, but fortunately it was decided to open the abdomen. We then found a large gall-bladder, dusky red in colour, and the wall much thickened. It was twisted on its pedicle, but had probably partly untwisted. The swelling was soft in consistency. There was a little free fluid in the abdomen. The gall-bladder was removed with surprising ease. After removal, it was opened, the wall was half an inch thick, the mucous membrane black. It contained one stone, the size of a pigeon's egg. The patient was not in the least upset by the operation and made a very quick and easy recovery.

Case 3 (R G P)—Mrs M M, aged 82, was admitted to the Bristol Royal Infirmary on March 25, 1933. It was quite impossible to obtain a lucid history from the patient as she was rather senile and almost completely deaf. The relatives, however, stated that the woman had complained of sudden severe epigastric pain that morning, accompanied by vomiting. The pain had been practically continuous, and the vomiting had recurred several times. There had been no movement of the bowels that day. No previous history of abdominal trouble was elicited.

The temperature was 100°, pulse 120 and irregular on admission. The patient was markedly shocked and the general condition bad. The abdomen was somewhat distended, and the right hypochondrium and iliac fossa rigid. Tenderness, which was present all over the right side, was most marked in the iliac fossa. No swelling was palpable owing to the muscular resistance, and there was no jaundice. Rectal examination was negative.

In spite of the patient's age, the diagnosis of acute perforative appendicitis was made, and the abdomen opened through a small incision in the iliac fossa. On incising the peritoneum a quantity of dark blood-stained fluid escaped, and the appendix was found to be normal. On exploring the right side of the abdomen with the fingers, a swollen tightly distended gall-bladder was found, which further examination revealed to be gangrenous. Rather than extend the incision, a second opening was made through the rectus below the right costal margin, and the gall-bladder, twisted on its pedicle through an angle of 180° in an anti-clockwise direction, was found and easily removed. In colour it was a deep purple-black, with an engorged wall $\frac{3}{8}$ in. thick. In addition to a little fluid, it contained a single cholesterol stone about the size of a marble. The abdomen was closed in the usual way and the patient made an uninterrupted recovery.

MECHANISM OF TORSION

In many recorded cases of torsion of the gall-bladder, the surgeon noted the presence of a mesentery. According to Brewer this is to be found in the anatomy department in 5 cases out of 100, and Ssulow confirms by reporting it 6 times in 145 cases. Our surgical experience would lead us to the opinion that it is nothing like so frequent as this, and text-books of anatomy, such as Poirier and Charpy, state that it is rare. Jacquement, quoted by Shipley, found it twice in 200 cases. It is probably a matter of nomenclature, obviously a part of the gall-bladder would be united to the liver by the mesentery far more often than the whole gall-bladder

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In our opinion a more important factor than a mesentery is a short attachment in our cases the cystic duct and the neck alone appeared to be attached to the liver, and the body and fundus were free. Probably in aged females the attachments have become loosened.

In three patients (Hansen's, Herfarth's, and Leriche's) the gall-bladder was hour-glass in shape, and only the distal portion was twisted.

The cause of the torsion is probably related to the movement of normal peristalsis in the transverse colon. As this sweeps across the colon three or four times a day from right to left it seems a probable suggestion that the lower surface of the gall-bladder might be carried along in an anti-clockwise direction, and it is interesting in this connection to observe that when the nature of the twist has been recorded, it has been anti-clockwise much more frequently than in the opposite direction.

One might have expected that stones in the gall-bladder would have been a predisposing factor by giving more purchase to the colon in producing a twist, but as a matter of fact stones are recorded as present in 15 cases and absent in 21 others. It is well known that in elderly women stones are frequently found, and the ratio is not much higher in the torsion cases, so this factor is evidently not important.

SIGNS AND SYMPTOMS

The great majority of patients have been elderly females, but a certain number have occurred in younger women. For instance, Daux's case was only 11, and Wendel's 23. There were 16 cases over seventy years of age. There have been only 6 cases in males, one of which was under our care. Previous history may show evidence of gall-bladder trouble. There may have been attacks of pain, and in one or two cases jaundice. On the other hand, many cases have enjoyed unbroken good health.

Signs and symptoms are fairly constant. The patient is seized rather suddenly with colicky pain in the centre of the abdomen or in the right hypochondrium which persists until operation is performed, although there may be remissions. Vomiting is usually present and sometimes urgent. Bowels may be constipated, normal, or there may even be a little diarrhoea. Jaundice is absent. For the first day or two pulse and temperature are normal, or only slightly raised (temperature 99°, pulse 90), but after two or three days both show marked rise. In most of the cases a definite rounded tumour, having the general characters of an enlarged gall-bladder, has been felt. It has often been recorded that the tumour has been under an anæsthetic, or not at all. There is a certain amount of tenderness and rigidity over the gall-bladder. As our second case shows, the tumour varies in hardness, it may be very easily felt on one examination and almost entirely gone a few hours later, owing, in all probability, to partial untwisting. This sign could scarcely occur in any other condition, except hydronephrosis, and perhaps intussusception.

After two or three days the condition of the patient deteriorates, and the gall-bladder has often been found gangrenous, though general peritonitis has supervened only in a few. The pulse and temperature rise, and delayed operation has usually failed to avert a fatality.

DIAGNOSIS

We believe that in those cases, the majority, in which a tumour having the general characters of an enlarged gall-bladder can be felt, a diagnosis can and should be made before operation, to the patient's very great advantage. The acute pain and vomiting, without jaundice, in an elderly female, and the appearance, *within a few hours of the onset*, of the greatly enlarged and palpable gall-bladder, are highly suggestive. If, as in one of our cases, and a few others (Brocq, Zum Busch), *the tumour comes and goes*, it can scarcely be anything else. Krabbel made a correct pre-operative diagnosis in his second case. We regret that we did not do so in ours. Kettner also correctly diagnosed his case before operation. The usual pre-operative diagnoses have been cholecystitis, hydrops or empyema of the gall-bladder (11 cases), intestinal obstruction (5 cases), appendicitis (7 cases), cancer of the colon or stomach (2 cases), perforated gastric ulcer (2 cases), twisted ovarian cyst (1 case), hydronephrosis (1 case).

TREATMENT

It is important not to delay operation in these cases on the supposition that it is a cholecystitis which will clear up. As the table shows, patients operated on after forty-eight hours often died, whilst all those treated early recovered. Surgeon after surgeon comments on the surprising ease with which the gall-bladder has been removed, the parts being so freely mobile. For this reason cholecystectomy is likely to lead to recovery even in patients of great age. Several, including two of ours, did very well though over eighty. In one case, Sutter's, the mobility of the parts led to the surgeon's undoing, for the ligature included the hepatic and common bile-ducts, and the patient died of obstructive jaundice.

Of the 39 cases operated on in which the result is stated, 34 lived and 5 died (operation on the third day in three, fifth and sixth one each). No doubt the true mortality is higher, fatal cases are apt to go unreported.

SUMMARY

1 Three cases of acute torsion of the gall-bladder are described, and a brief report is given of fifty others from the literature.

2 A pre-operative diagnosis could often be made, the grounds being the occurrence of acute pain and vomiting, especially in an elderly female, with a palpable tumour having the general characteristics of an enlarged gall-bladder, which comes up within a few hours, and may appear and disappear. In some cases, however, the swelling is masked by rigidity and tenderness.

3 Early cholecystectomy usually saves the patient, even in octogenarians. The operation is a very easy one in these cases.

4 Cases of recurrent partial torsion are on record, but are less frequently seen.

BIBLIOGRAPHY

- FISCHER, *Zentralb. f. Chir.*, 1925, lii, 1527
 KRUKENBERG, *Berl. klin. Woch.*, 1903, July, 667
 BREWER, *Ann. of Surg.*, 1899, xxi, 721

- SSULOW, Quoted by Fischer
 WENDEL, *Ann of Surg*, 1898, xxvii, 199
 MAYER, *Berl klin Woch*, 1908, xiv, 1602
 MUHSAM, *Ibid* 1179
 NEHRKORN, *Dent Zeits f Chir*, 1908, xcvi, 319
 LETT, *Lancet*, 1909, 1, and *Brit Jour Surg*, 1921-2, ix, 464
 FISCHER, *Berl klin Woch*, 1910, xlvii, 1784
 KUBIG, *Munch med Woch*, 1912, lix, 1999
 CRAMP, *Med Record*, 1915, Jan 16, 120
 HENSCHEN, *Schweiz Aerzt*, 1915, xiv, 502
 REICHEL, *Munch med Woch*, 1919, Aug 1, 884
 KRABEL, *Dent Zeits f Chir*, 1920, 76
 STRAUSS, *Beitr z klin Chir*, 1921, xxi, 519
 HANSEN, *Hospitalstidende*, 1921, March 30
 FRANKAU, *Brit Jour Surg*, 1922-3, x, 301
 IRWIN, *Ibid*, 1921-2, ix, 310
 JONAS, *Brit Med Jour*, 1923, 1, 1016
 DAUX, *These de Paris*, 1924
 LUTAUD, *Soc Nat de Chir*, 1924
 DUBS, *Schweiz med Woch*, 1924, Jan, 93
 FIFIELD, *Brit Med Jour*, 1925, 1, 920
 HUDDY, *Lancet*, 1926, ii, 120
 GRUNERT, *Arch f klin Chir*, 1927, cxlvii
 SUTTER, *Beitr z klin Chir*, 1925, March, 519
 RAMSHORST, *Nederl Tijds v Geneesk*, 1926 Dec, 2798
 CECCARELLI, Quoted by Costantini
 DARAGNEZ, *Soc Nat de Chir*, 1926
 FELDMAN, *Schweiz med Woch*, 1926, ii 35
 MARINACCI, S, *Polichinico*, 1926, xxxiii, 194
 ESAU, *Arch f klin Chir*, 1927, cxlvii
 ZUM BUSCH, *Munch med Woch*, 1927, lxxiv, 1099
 HOLDEN, *Jour Amer Med Assoc*, 1927, April, 1077
 SHIPLEY, *Arch of Surg*, 1927, May, 968
 ROMANI, *Minerva Med*, 1928, Sept, 660
 MEADE, *Brit Med Jour*, 1929, 1, 687
 GWYNNE, *Ibid*, 1930, 1, 823
 COSTANTINI, *Polichinico*, 1930, xxxvii, 149
 KETTNER, *Zentralb f Chir*, 1930, lvii, 589
 BROCCQ, *Presse med*, 1931, July, 1015
 KOWALENSKI, *Dent Zeits f Chir*, 1931, 809
 MURRAY, *Brit Jour Surg*, 1932-3, xv, 687
 MEER and LISERBY, *Southern Med Jour*, 1932, June, 609
 ROUHIER and LERICHE, *Soc Nat de Chir*, 1932, July, 1045
 HERFARTH, *Zentralb f Chir*, 1932, Sept, 2268

ONE-STAGE LOBECTOMY FOR BRONCHIECTASIS* AN ACCOUNT OF FORTY-EIGHT CASES

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ALL writers on pulmonary disease are agreed that bronchiectasis is a pathological condition of which the progress is only delayed by intermittent palliative measures. As is stated by Powell and Hartley, "Bronchiectasis, owing to its anatomical characters, is not a disease from which we may expect recovery. At the best, all that we may hope for is that by preventing decomposition of the sputum, we may be able to arrest its further progress. In nearly all cases, in spite of treatment, the disease gradually advances, although its duration varies much in different individuals, in some cases being exceedingly chronic, in others running a comparatively acute course." It is for this reason that attempts have been made by operative measures to arrest or to eradicate the disease. Necessarily, operations upon individuals who have been for some considerable period subjected to the results of chronic infective processes assume a severity which would not be so marked a feature in a relatively healthy individual.

PATHOLOGY AND SYMPTOMATOLOGY OF BRONCHIECTASIS

It is not our intention here to discuss otherwise than very broadly the etiology and pathology of bronchiectasis, for its causation is still a matter of some doubt and the rate of its progress varies considerably in different individuals. Unquestionably there is a condition of primary bronchiectasis—i.e., a dilatation of the bronchi unassociated with infection or any other gross pathological lesion in the lung. This has been described as congenital, and arises primarily in the lower lobe, more particularly in that of the left side. Whether this type invariably becomes secondarily infected and gives rise to symptoms is debatable, but that a certain proportion of these cases do so is unquestionable.

Secondary bronchiectasis may arise as a sequel to infective processes, such as bronchitis, lung abscess, tuberculosis, etc., and to conditions causing intermittent or more permanent obstruction of a greater or lesser portion of the bronchial tree, such as asthma, foreign body, or new growth, innocent or malignant. Bronchiectasis also arises secondary to lung abscesses and empyemata, especially those in which fibrosis extends into the lung.

The addition of an infective process in either the primary or secondary varieties leads to more rapid progress of the disease and results in further weakening and disorganization of the structures composing the walls of the bronchi. This

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may result in considerable deposit of fibrous tissue in the bronchial walls when the infection is subacute or chronic, whereas, in the more acute types the spread may occur into the adjacent lung, causing abscess formation or even gangrene. Further, in chronic cases, persistent presence of infective secretions in the bronchial tubes is liable to initiate repeated attacks of broncho-pneumonia and often results in the spread of the disease to adjacent portions of the same lung or the base of the opposite lung.

Ulceration of the mucous membrane of the bronchi or the excessive formation of granulation tissue may cause hæmoptysis, which is commonly of the intermittent type, but sometimes results in rapid death from a large hæmorrhage.

Symptoms and signs are so well recognized as not to merit description here.

TREATMENT

As an indication of the proportion of patients with unilateral or unilobar bronchiectasis Jex-Blake, analysing the autopsy findings in 105 patients, has shown that the disease was still unilateral in 61 and confined to one lobe in 34 patients. This shows the high proportion of patients, subjects of this disease, who could, from the localization of the disease, have been candidates for some form of surgical procedure. The immediate cause of death was bronchopneumonia and exhaustion in 76 patients, hæmoptysis in 5, and cerebral or cerebellar abscess in 15. The high proportion of brain abscesses is significant, particularly as operative procedures have so often in the past been held responsible for this fatal complication, and in view of the fact that none of these patients had been submitted to any surgical procedure.

A very brief resume of the methods of treatment which have been adopted at various times will give some indication of the value of these different measures.

NON-OPERATIVE PROCEDURES

1 **Drugs.**—Simple medical treatment consists of rest and the use of certain drugs intravenously, by inhalation, or by mouth. These consist of arsenical compounds, given by intravenous or intramuscular injection, for their specific effect on spirochetes, which are not rarely found in the sputum in this disease, lipiodol, which is generally given into the bronchi by intratracheal instillation, and such other drugs as emetine and creosote given by mouth. It is very doubtful if the effect of any of these is more than temporary, and no claim has ever been substantiated as to their permanent curative value.

2 **Postural Drainage.**—Temporary improvement always results from such measures as postural drainage, in which the patient is placed in such a position, found after trial, as will most easily empty the secretions in the bronchi. The position is taken up morning and evening and in many cases will enhance the patient's comfort during the intervening periods.

3 **Bronchoscopic Aspiration**—This also is of considerable temporary value, often improving the general condition of the patient to a marked degree. The value of instillations of antiseptics through the bronchoscope is doubtful. In the presence of a foreign body or of stenosis, the value of bronchoscopy is unquestionable. It must be remembered, however, that removal of a foreign body which has been *in situ* in a bronchus for a considerable period and has caused gross

dilatation of the tubes in the obstructed portion of the lung will not necessarily lead to recovery. In other cases bronchoscopy can only be regarded as a more efficient form of drainage than that of posture, and any fresh infection will tend to light up the process in the involved area of the lung again. Radiological examination after lipiodol injection, following a course of bronchoscopy, in nearly all except very early cases shows no diminution in the dilatation.

OPERATIVE PROCEDURES

Operative measures may be divided into two groups (1) Collapse methods, and (2) Radical extirpation of the diseased tissues.

I. COLLAPSE METHODS

All collapse measures have certain disadvantages due to the fact that the diseased tissue still remains, but it is possible that the activity of the infection may be kept in check in many cases by the approximation of the bronchial walls and the avoidance thereby of stagnation, while the compression is maintained.

Artificial Pneumothorax is the most simple method of general compression of the lung, and has been used freely in the treatment of bronchiectasis. In early cases, and especially where the bronchial walls are relatively soft, it may prove of considerable value, but it is unlikely, even when pleural adhesions do not prevent the establishment of pneumothorax, that it could collapse the thickened, rigid, dilated bronchi found in the well-established disease. In fact, the mediastinum is much more likely to be displaced than the bronchi collapsed if the pressure of the air within the pleura is increased.

In cases in which, as a result of artificial pneumothorax, the sputum has almost disappeared, when the pneumothorax is discontinued it is not uncommon to see the expectoration increasing again *pari passu* with the expansion of the lung. Further, prolonged maintenance of an artificial pneumothorax is often impossible owing to the development of a pleural effusion or to the pneumothorax cavity contracting in spite of regular refills with air.

Oleoathorax, or the replacement of the air by sterile oil, is claimed by some authorities to overcome some of these objections, but the proportion of patients in which permanent improvement can be expected by this method is small, and the oil has the added disadvantage of causing dense pleural thickening, which may prevent any subsequent radical operation, or, at the least, the post-operative expansion of the remaining lobe.

Phrenicectomy has been extensively used for bronchiectasis, with varying results. There is no doubt that in early bronchiectasis—for example, that type secondary to basal pulmonary abscess—all symptoms can be made to disappear permanently. In other cases no change is to be noted, and occasionally the operation may result in angulation of the bronchi and cause interference with the drainage. Examination of the diaphragmatic movement under the X-ray screen in cases of bronchiectasis almost invariably shows limited mobility due to spasm induced by irritation of the infected neighbouring lobe. This may be the cause of poor evacuation of secretions from the lower bronchial tubes, in many cases diaphragmatic paralysis will diminish dyspnoea due to this cause and will allow the unimpeded contraction of the powerful abdominal muscles during cough to

result in more complete evacuation of the contents of the dilated basal tubes. Thus dyspnoea on exertion may be less evident, sputum and fœtor be diminished, and definite improvement follow.

Thoracoplasty has been widely used for bronchiectasis, and the results appear to be variable. It is unlikely that the relatively restricted thoracoplasty used by Sauerbruch for tuberculosis could produce sufficient collapse to free patients of symptoms. Hedblom and others have advocated wide removal of the ribs almost to the extent of complete decostalization by multiple-stage operations. By this more radical procedure he claims a fair proportion of success, but, as is mentioned earlier, the results cannot be expected to be so satisfactory as those following removal of the diseased area. However, in cases of almost complete unilateral bronchiectasis thoracoplasty is still the operation of choice and gives a definite probability of considerable amelioration. Its value where the disease is localized is problematical. Thoracoplasty should invariably be performed in two or more stages, preceded by a phrenicectomy.

Pre-operative treatment should be carried out in the same way as for lobectomy.

2. RADICAL OPERATIONS

Cautery Pneumectomy was originally devised by Graham with the idea of removal of the diseased portion of the lung by repeated cauterizations. The value of the operation appears to be largely due to the production of efficient drainage in suppurative conditions of the lung. In definite cases of bronchiectasis unassociated with the formation of large cavities, we have not found this operation satisfactory. Its author now advises its use in bronchiectasis associated with multiple lung abscesses and in chronic lung abscess with secondary bronchiectasis. In those cases secondary to foreign-body inhalations, especially if associated with bronchopleural fistula or empyema, and who are suffering from the effects of prolonged sepsis, the pulmonary drainage produced by cauterization may tide them over a critical period. Summing up the value of cautery pneumectomy, Graham now states that "the operation can be recommended to those suffering from unilateral bronchiectasis who have not responded well to the simpler forms of therapy, for whom thoracoplasty is either not indicated for various reasons or has failed to relieve the symptoms and upon whom the performance of a lobectomy, although desirable, is unwise or technically impossible."

One-stage Lobectomy is dealt with fully below.

ONE-STAGE LOBECTOMY FOR BRONCHIECTASIS

Removal of a lobe of lung the site of bronchiectasis has been the ideal aim of surgical treatment for many years, and it is surprising to find in how large a proportion of patients the disease may be relatively advanced in a single lobe without gross disease in other parts of the lung. When the disease is confined within these limits the removal of the infected area should cure the patient if he survives operation and his immediate post-operative period.

Fear of widespread infection of the pleura and a mobile mediastinum have deterred the majority of thoracic surgeons from attempting one-stage lobectomy. As a result operations with two or more stages were performed, the earlier stages

with the object of closing off the upper part of the chest by adhesions and causing thickening of the pleura in the neighbourhood of the mediastinum. The largest series in this group was published by Lilienthal, with forty-two cases and a mortality of 64·3 per cent. Alexander, using a different technique, has much improved the results, with a mortality of 16·6 per cent in his first twelve cases, but this operation entails removal of several ribs, with a subsequent deformity of the chest.

The one-stage method was first tried out in a short series of eight cases by Brunn, and later slightly modified by Shenstone. Most one-stage lobectomies have been performed on these same principles.

DIAGNOSIS AND INDICATIONS

It is essential to ensure that the disease is localized to one lobe or to the middle and lower lobes on the right side. This can be definitely determined by radiological examination following lipiodol intratracheally, but this examination should be carried out not only antero-posteriorly but laterally, and if necessary obliquely as well, and of course both lungs should be examined. In one case in this series when this was impossible owing to preliminary treatment by pneumothorax, the disease has been shown subsequently to involve the lower portion of the upper lobe.

It does not appear generally justifiable to advise lobectomy where definite disease can be demonstrated in the opposite lung, as there is not only a greater post-operative risk but there is a great probability of the disease extending. In unilobar bronchiectasis there appear to be two main groups in which to advise operation: (1) Patients who expectorate definite quantities of pus daily, especially when it is offensive in odour, and (2) Patients who have repeated hæmoptyses with or without offensive sputum. The latter group are more favourable as infection is usually less intense. Those in whom bronchiectasis is secondary to neglected pulmonary abscess are also suitable, but when empyema, whether primary or secondary, has occurred, the operation may offer considerable technical difficulty from firm pleural adhesions.

Repeated examination of the sputum should be carried out to eliminate the presence of tubercle bacilli.

It is probable that lobectomy will be recognized in the near future as the ideal treatment for the thick-walled chronic lung abscess, especially those associated with localized bronchiectasis.

PRE-OPERATIVE TREATMENT

The preparation of the patient is of very considerable importance, and steps should be taken to raise the local and general resistance to the greatest possible degree, this is attained by the following measures:

1 **Postural Drainage**—The regular adoption of postural drainage unquestionably improves the condition of the patient by diminishing the quantity of the sputum and, by the improved drainage, the virulence of the organisms in the purulent discharge. A similar result can be obtained by placing the patient regularly in a creosote chamber, which induces deep coughing. These methods may be combined.

2 **Preliminary Bronchoscopy**—Aspiration via the bronchoscope is certainly more effective than postural drainage, but, although a minor procedure, is not so well tolerated as a pre-operative measure

3 **Vaccine Therapy**—In the great majority of these cases a vaccine has been prepared from the sputum and each patient has been given three increasing doses at weekly intervals before operation. The value of this measure is difficult to assess, but it is possible that it raises the patient's resistance and may help thereby to limit the effect of aspiration into the other lobes and to increase pleural resistance

4 **Nose and Mouth Examination**—Complete examination of the upper air-passages, including sinuses and teeth, is essential, and any obvious focus of infection should be eradicated by suitable treatment

5 **Artificial Pneumothorax**—In all cases where extensive pleural adhesions do not prevent its accomplishment, artificial pneumothorax is induced one week before operation. This is done for two purposes (*a*) There is much less pulmonary movement when the pleura is opened and therefore less upset to the patient and anæsthetist, and (*b*) It tends to prevent accumulation of secretions in the affected lobe up to the time of operation. (If pneumothorax is produced only a few days before the major procedure, it neither prevents rapid expansion of the remaining portion of lung nor does it cause pleural thickening)

6 **Phrenic Nerve Operations**—No procedure is carried out on the phrenic nerve either before or at operation. Although temporary paralysis of the diaphragm done as a preliminary may make the operation technically easier, the movements of the diaphragm are of considerable importance in aiding expansion of the remaining lobe in the post-operative period, and its paralysis can conceivably delay this to a definite degree. Moreover, crushing at the time of operation may open up a passage for organisms through the mediastinal pleura. In cases in which the remaining upper lobe is firmly attached by adhesions, a late phrenic evulsion will assist in closing a residual basal empyema, and is reserved, for this purpose, to those cases requiring it

7 **General Preparation**—This consists of the administration of glucose by mouth for several days and, in addition, per rectum for the day preceeding operation. Furthermore, it is a wise precaution to group the patient's blood in preparation for a possible transfusion

ANÆSTHESIA

(By Dr IVAN W MAGILL)

Certain points require special consideration in lobectomy

1 **Preliminary Medication**—This should be sufficient to ensure the comfort of the patient and facilitate induction without incurring respiratory depression in the post-operative period. The most satisfactory combination is a suitable dose of omnopon (up to $\frac{1}{3}$ gr) and scopolamine (up to $\frac{1}{150}$ gr) administered forty minutes before operation, followed by a minimum basal hypnotic dose of a quick-acting barbiturate, such as nembutal or evipan. The barbiturate is given intravenously just before induction

2 **The Anæsthetic**—Rapidly of elimination and absence of toxic effects make nitrous oxide and oxygen the anæsthesia of choice. Should any supplementary

agent be required, chloroform alone is permissible, owing to the use of diathermy. For the same reason, ether, ethylene, or other inflammable agents are contra-indicated.

3 Method of Administration—Any efficient gas-oxygen machine can be used. A water manometer on the apparatus is an advantage, as it is more sensitive than the mercurial or mechanical type of pressure gauge. The anæsthetic can be administered throughout with a close-fitting facepiece, but this procedure involves certain risks. (a) The patient lies on the sound side and, in this position, unless constant positive pressure is maintained, secretions may be dislodged into the trachea and thence into the sound lung during manipulation of the affected lobe, (b) Unless an undesirable depth of anæsthesia is maintained, reflex closure of the glottis may occur during traction on the pulmonary root. Both these risks can be eliminated by intubation anæsthesia combined with partial occlusion of the bronchus on the affected side by means of a coude gum-elastic catheter.

For intubation, rubber tubes are used, the calibre being sufficient to permit to-and-fro breathing through the lumen. When no contra-indication exists, the rubber tube is passed through the nose owing to the ease with which this manœuvre can be carried out in the majority of cases. Anæsthesia being established, the coude catheter is passed through the glottis with the aid of a laryngoscope and stilette. To ensure entry into the bronchus, the tip of the catheter is kept in close contact with the lateral wall of the trachea by means of the stilette. When practicable, this form of anæsthesia provides the safest operating conditions. (a) On opening the pleural cavity, when the lung is free it can be seen to be deflated, and is therefore more easy to handle, (b) Secretions can be removed through the catheter by suction, as often as necessary, (c) Continuous positive pressure is not essential, and on this account there is less impediment to the right side of the heart.

OPERATIVE TECHNIQUE

Incision—A curved incision is made overlying the seventh interspace and curving upwards posteriorly. The muscular layers are divided similarly to the mid-axillary line. When the ribs are exposed an incision is made in the intercostal space, a small Tuffier retractor inserted, and the incision widened. Posteriorly, the seventh rib is divided subperiosteally behind the angle, and a segment of rib $\frac{3}{4}$ in removed. (The removal of this small portion, rather than division only, definitely limits post-operative pain.) The intercostal vessels are then ligatured in two places, and the incision carried backwards between the ligatures and forward to the mid-axillary line.

It is essential, in view of the highly infective nature of the contents of these infected lobes, to pay particular attention to the protection of the muscular chest-wall. In addition, therefore, to the application of tetra cloths to the skin as soon as the pleural cavity is exposed, the whole of the opening is lined with gauze saturated with 1-1000 flavine, which is retained in position for the remainder of the operation. Wide retraction is now carried out.

In a large proportion of cases adhesions are present, most commonly over the diaphragm, but in many cases posteriorly and between the lobes. In some cases adhesions are generalized, and in many cases very dense. (In two cases the attachments of the lobe to the lower posterior part of the chest were so dense and vascular that it was necessary to secure the hilum before they were cut.)

When the adventitious adhesions are all freed, the lobe is lifted upwards and the pulmonary ligament is exposed. In almost every case this will be found to contain vessels of appreciable size, and it should therefore be divided between forceps. Ligatures are then applied and the lobe is free and ready for control of the pedicle. Occasionally the normal interlobar space may be absent or partially defined, and in these cases it is necessary to divide the bridge of lung tissue, the raw area on the remaining lobe being sutured later.

Removal of the Lobe.—Before application of the lobectomy tourniquet (*Fig 193*) it is essential to pack off the pleura carefully to avoid soiling when the

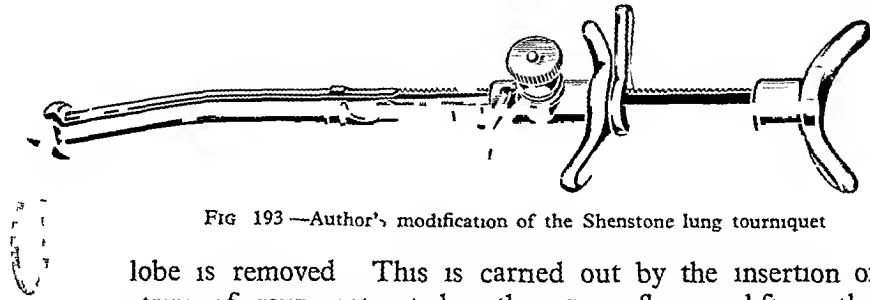


FIG 193 —Author's, modification of the Shenstone lung tourniquet

lobe is removed. This is carried out by the insertion of long strips of gauze saturated with 1-1000 flavine, lifting the lobe to be removed by holding forceps while this is accomplished. A strip of gauze that has been wrung out in 10 per cent cocaine is now wrapped around the hilum of the affected lobe for a short time to anæsthetize nerve-endings there and prevent reflex effects. The loop of the tourniquet is dropped over the

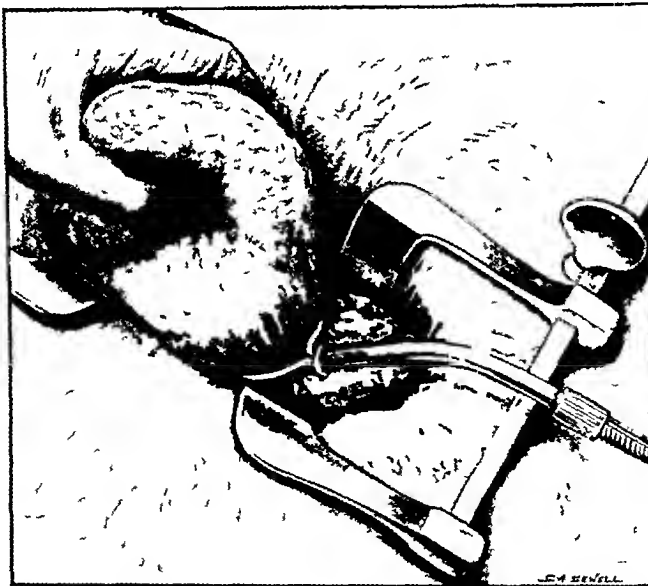


FIG 194 —Placing of tourniquet around lobe, to be followed by packing off remainder of chest

lobe and adjusted accurately to the hilum (*Fig 194*). The tension on the silk can be accurately determined before it is locked in position. In our experience the application of a second tourniquet is either impossible or unnecessary. In

those cases in which there may be much retention of purulent secretions in the bronchi, the lobe is generally found to be solid and therefore the application of a second tourniquet is impossible, whereas in those cases in which the second tourniquet can be applied, the contents of the bronchi are minimal and its application is unnecessary. Careful swabbing during the division of the hilum is always essential.

The hilum is divided distal to the tourniquet with scissors, the lobe removed, and any projecting bronchi are cut short. The division at the hilum was carried out by diathermy in the early cases, but this does not appear to have any advantages and may result in necrosis of the cartilage of the bronchial stumps. Novarsenobenzol in solution is applied to the stump in order to destroy any anaerobic organisms which may be present. The lumina of the main bronchial divisions in the stump are carefully swabbed with pure carbolic or 30 per cent silver nitrate to destroy the mucous membrane.

Suture of Stump—The stump controlled by the tourniquet is now sutured by the insertion of a series of mattress sutures (*Fig 195*). It is advisable to use

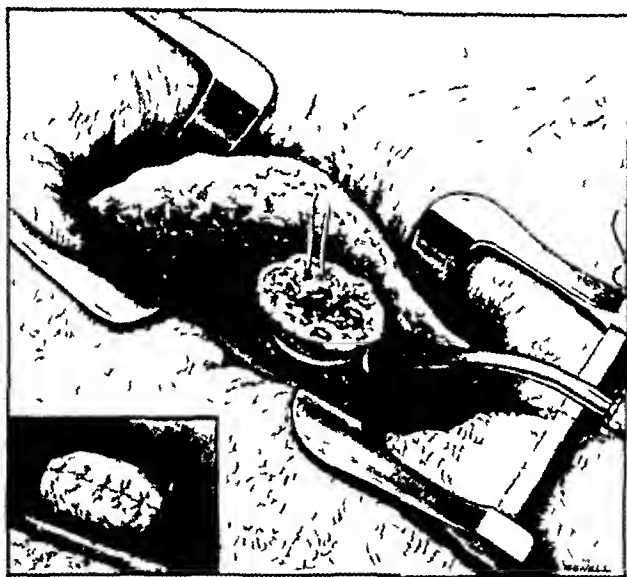


FIG 195—Method of suturing stump by mattress sutures closing bronchi and sealing vessels. Inset shows suture of edges over bare area of stump.

chromic gut with a needle at each end of the suture, and to commence the sutures at the lower end. The ends of these sutures are kept long and held in forceps until the whole surface of the stump is controlled. The lock on the tourniquet is released and the tension gradually diminished by the assistant. On signs of any bleeding the tension is increased while the area is controlled by the insertion of another suture. When the stump is secure the tourniquet is removed and the stump oversewn, where possible, by the fringe of pulmonary tissue.

In the majority of cases no attempt has been made to attach the stump to the adjacent lobe owing to the possibility of infecting this from the passage of sutures through the septic pedicle into the normal lung above, and if left alone there is

every probability of rapid adhesion from expansion of the upper lobe over the stump

Positive pressure induced through the intratracheal catheter or the induction of cough will disclose any leakage in the stump, which when present is controlled by further suture

Closure of Chest—This is attained by drawing the ribs together by three or four pericostal sutures passed through the middle of the interspace above and below the incision to avoid damage to the intercostal vessels and nerves. The intercostal muscles and the deep and superficial muscles of the chest wall are sutured with continuous catgut. Three or four deep tension stitches of strong silkworm gut are inserted, and the skin closed by continuous silk.

Drainage.—In the first case in this series (1929) in which lobectomy was performed, without any form of tourniquet on the stump, the chest was closed completely. Subsequently collections of fluid without gross infection were aspirated on ten occasions. Later, following Shenstone's technique, an intercostal tube was invariably inserted upwards to the stump through a cannula introduced below the main incision. In the course of these cases it was often found that there was considerable difficulty in getting the intercostal tubes to function adequately, and in many cases collections of fluid, often sterile, have had to be aspirated from different pockets in the pleura. The essential to be attained in the early stage is full expansion and adhesion of the upper lobe, and it seemed to us that the value of the intercostal tube was doubtful. The second condition which appeared to call for immediate drainage is that of high-tension pneumothorax should the stump leak. This risk again, from our experience, we believe to be exaggerated. If the stump leaks in the first twenty-four hours there will be definite evidence of surgical emphysema to warn those in attendance of the fact that air is escaping into the pleura, for it is quite impossible to suture the chest sufficiently firmly to prevent the escape of air under tension from the pleura to the extrathoracic tissues. Leakage from a bronchial fistula at a later stage does not offer any serious risk, as it is only into the area around the stump and is shown by the patient's coughing up pus from a residual cavity around the stump. Likewise, in view of the modern treatment of extensive infected pleural effusions by aspiration in the early stages—i.e., before adhesions have formed—it appeared reasonable to treat these patients on similar lines.

In six cases treated on these lines the chest wound was completely closed. Either in the operating theatre or immediately on the patient's return to bed, a needle attached to a pneumothorax apparatus was inserted into one of the upper interspaces anteriorly and as much air as could be obtained was aspirated. Estimation of the intrapleural pressure immediately disclosed any leakage from the lung. The one disadvantage of this method was that it necessitated repeated and early aspiration of the chest. This had a disturbing and wearing effect upon the patient, and latterly we have again altered the technique by introducing an intercostal tube which is closed by a spigot, then aspirating the air from the upper pleura with a pneumothorax apparatus.

When the patient has recovered consciousness in bed, the tube is connected with an underwater tube, care being taken to allow no air to enter the chest during this procedure, and fluid from the pleura can be evacuated by releasing the controlling clip as and when required.

The essential factor is to get full expansion and adhesion of the remaining lobe before evidence of infection shows itself, so that, subsequently, the most serious eventuality is a localized empyema cavity

The aspiration of air from the upper chest immediately after operation appears to result in more even expansion of the upper lobe and less liability to the formation of residual pleural pockets requiring aspiration

POST-OPERATIVE TREATMENT

Blood Transfusion may be required in some cases, and although the necessity appears to diminish with increasing experience of the operation it still depends to a large degree upon the pathological conditions found. Thus, for cases with extensive and firm adhesions which result in a considerable degree of oozing from the chest wall, blood transfusions are more often required, and as it is impossible to determine these with certainty beforehand, every patient should be grouped as a pre-operative measure (N.B.—Although the production of pneumothorax will determine the presence of wide adhesions, dense adhesions are occasionally found in the costo-vertebral groove and to the pericardium, which may not be disclosed by X-ray examination)

In every case there will be a good deal of blood-stained discharge from the pleura in the course of the two days following operation, and in all cases in which there would appear any doubt about the immediate necessity of post-operative transfusion it should be performed

General Post-Operative Measures—The usual routine administration of fluids—per rectum, subcutaneously, or intravenously—is carried out according to the necessities of the case. Cardiac stimulants, coramine, cardiazol, etc., are administered when necessary, and occasionally the respiratory tract may require stimulation by the administration of CO₂ and oxygen

The patient on recovery from the effects of the operation and anæsthetic is nursed in the sitting posture to allow free use of the lower intercostal muscles and diaphragm

The Condition of Pleura at Operation—In 38 patients the pleura has been free at the time of operation—i.e., the remaining lobe was not adherent to the chest-wall. In 17 of these patients the affected lobe had definite adhesions, varying from those in which the adhesions could be separated by blunt dissection to those which required division with sharp instruments. In the remainder adhesions were generalized over all lobes, varying considerably in density

Bronchial Fistula—The formation of a bronchial fistula is, in our experience, common, and usually occurs on about the tenth day. It is not as a rule of any particular significance, for if the intercostal tube is still draining no risk is entailed, and if the chest-wall has healed it results in the expectoration suddenly and temporarily increasing in quantity from evacuation of the small residual cavity around the stump

Treatment of Late Empyema.—The residual infected cavity usually heals without further drainage than that established by the intercostal tube, but in a few severely infected cases rib-resection may be necessary. By the time that this is required, the remaining lobe or lobes are adherent to the chest-wall, the only

complication being the presence of the bronchial fistula. The bronchial opening, being deep, tends to close spontaneously as the cavity contracts, and in only one patient has the fistula persisted over three months.

Late Phrenicectomy—This was performed in eight cases in order to close the residual cavity and bronchial fistula, and was successful in its effect in seven patients.

ANALYSIS OF CASES (*see Table, pp 322-325*)

This series consists of 48 consecutive cases, of whom 23 were men and 25 women. The disease occurred in the lower lobes in 47, and in the upper lobe in 1 patient. The lesion was situated in the right lung in 14 patients, was bilateral in 1, and in the remainder was situated on the left side. In two patients the right middle and lower lobes were involved and required resection.

Six patients were over 40 years of age, the youngest was 9 and the oldest 49 years.

Predominant Clinical Features—Hæmorrhage was the predominant symptom in 11 patients, whereas in the remainder fetid purulent expectoration was the chief indication for operative procedure, although hæmoptysis accompanied this symptom in a further 20 cases.

Post-operative Drainage.—This was carried out by intercostal tube in 42 cases. No post-operative drainage was carried out in 6 patients. Of these, 4 healed per primam, following aspiration, although in a few other drained cases intercostal drainage ceased early, the intercostal tube was removed, and the effusion was aspirated subsequently. In no case has the main wound broken down and allowed free communication between the pleura and the exterior. In the great majority the main wound has healed per primam.

Mortality—Seven patients have died subsequent to lobectomy, four within the first week and three later. Of the four early post-operative deaths, three succumbed to shock and hæmorrhage and one to suppurative bronchitis of the opposite lung. Of the three late deaths, one resulted from cerebral abscess four weeks after operation when the local condition consisted of a small residual sinus, and of the other two, one three months after operation from a spontaneous pneumothorax due to unsuspected tuberculosis on the unoperated side. This latter patient, however, still had a large empyema cavity remaining on the operated side. The third, a case of bilateral disease, died five months after operation with a residual empyema, tuberculosis of the opposite lung, and a terminal streptococcal effusion in the opposite pleura and pericardium.

Analysis of the Surviving Patients.—These number 41, of whom 6 are still in hospital and convalescent. Excluding these, there remain 35 patients who have been discharged from hospital at varying periods from five years to a few weeks, and, with the exception of one patient, are completely healed. This patient still has a small residual empyema cavity with a persistent bronchial fistula of over a year's duration, and may later require a plastic operation for its closure. Six of the remaining 34 patients still have some residual symptoms, all of much diminished degree. In one, in whom the operation was performed for bronchiectasis associated with a sinus in the chest-wall and in whom repeated severe hæmorrhage was the indication for surgical interference, there has been a recurrence of a minor degree

ANALYSIS OF CASES OF BRONCHIECTASIS TREATED BY ONE-STAGE LOBECTOMY

NAME	SEX AND AGE	SIDE	SITE	EXPECTORATION	HEMOPHTYSIS	DATE OF OPERATION	STATE OF PLEURA	DRAINAGE	LATE PHRENIC-ECTOMY	REMARKS
1 A L	F 17	L	Lower	30-60 cc	60-90 cc	17 4 29	Pleura free adhesions	Intercostal	No	No symptoms
2 M D	F 32	L	Lower	Nil	Severe repeated	29 3 30	Densely adherent	Intercostal	Yes	Quite fit and well Occasional small hamorrhages followed by expectoration of thread suture from stump
3 G T	M 19	L	Lower Adhesions	360-450 cc	Nil	12 2 31	Adhesions to diaphragm and pericardium Upper lobe free	Intercostal	Yes	20 4 34 purulent sputum daily, but working
4 L Y	M 24	L	Lower	60 cc	120 cc	25 2 31	Dense adhesions to upper lobe Old A.P. Upper lobe free	Intercostal	No	No symptoms
5 A B	M 21	L	Lower	60-90 cc	Considerable	22 4 31	Pleura free	Intercostal	No	Has recently developed T.B. 20 4 34
6 E M	F 17	L	Lower	300-450 cc	Nil	3 6 31	Lower lobe densely adherent to upper Both lobes	Intercostal	No	No symptoms Gained 1 stone
7 E S	M 30	L	Lower	150-210 cc	Severe persistent	1 7 31	Densely adherent	Intercostal	Yes	25 4 34 Died 2 7 31 Cardiac failure
8 J H	F 14	L	Lower	30-60 cc	Recurrent	4 7 31	Dense adhesions throughout	Intercostal	Yes	No symptoms Gained 1 1/2 stone
9 W E	M 24	L	Lower	120 cc Purulent	Nil	2 3 32	Diaphragmatic adhesions Pleura free	Intercostal	No	No symptoms
10 E S	M 24	L	Lower	240 cc	Copious	28 7 32	Dense adhesions of lower and upper lobes	Intercostal (1) Secondary (2)	No	No symptoms Gained 1 stone
11 B H	F 31	L	Lower	60 cc	Repeated	24 8 32	Diaphragmatic adhesions Upper lobe free	Intercostal (1) Secondary (2)	No	Died 28 9 32 Aortic abscess R. temporary R. temporary Small bronchial fistulous tract to skin No symptoms
12 J M	M 9	R	Middle and lower	30 cc	600-1200 cc	7 9 32	Moderate adhesions Upper lobe	Intercostal	No	No symptoms

LOBECTOMY FOR BRONCHIECTASIS

Case No.	Sex	Age	Site	Size	Character	Adhesions	Pleura	Operation	Result	Remarks
14	W	G	R	45	Lower	60-90 cc Purulent 15 cc	Nil	27 12 32	Pleura free	Intercostal
15	E	H	L	36	Upper	Repeated at- tacks of pneu- monia 40-60 cc Fœtid	Small 180 cc	25 1 33 22 2 33	Both lobes ad- herent Dense adhesions of both lobes	Intercostal
16	D	H	L	16	Lower		Once	15 3 33	Incomplete fis- sure, otherwise free	Intercostal
17	E	G	L	38	Lower Cylindrical Saccular		Nil	22 3 33	Both lobes dense- ly adherent	Intercostal (1) Secondary (2)
18	C	R	R	29	Lower	240 cc	Nil	5 4 33	Pleura free	Intercostal
19	J	D	R	21	Lower	30 cc	Occasional	10 4 33	All lobes densely adherent	Intercostal
20	J	J	R	31	Cavities middle and lower lobes	800 cc	Nil	13 5 33	Pleura free	Intercostal (1) Secondary (2)
21	E	M	R	34	Lower	90 cc	Nil	8 6 33	Pleura free	Intercostal (1) Secondary (2)
22	B	T	L	20	Lower	30-60 cc	Small			Intercostal
23	W	T	R	11	Lower	Intermittent	Nil	10 7 33	Affected lobe ad- herent to sinus, otherwise free	Intercostal
24	T	D	L	38	Lower	180-360 cc	Nil	12 7 33	Pleura free	Intercostal
25	I	C	L	30	Lower	120 cc	Up to 300 cc	26 7 33	Pleura free	Intercostal

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ANALYSIS OF CASES OF BRONCHIECTASIS TREATED BY ONE-STAGE LOBECTOMY—continued

NAME	SEX AND AGE	SIDE	SITE	EXPECTORATION	HEMOPTYSIS	DATE OF OPERATION	STATE OF PLEURA	DRAINAGE	LATE PHRENIC-ECTOMY	REMARKS
26 E M	M 15	L	Lower	120 c c	30 c c	2 8 33	Pleura free	Intercostal	No	No symptoms Trace of sputum 2 5 34
27 C J	F 33	L	Lower	30 c c	Repeated	23 8 33	Pleura free	Intercostal	No	No symptoms 6 c c mucoid spu- tum daily Other- wise well 4 4 34
28 G C	F 17	R	Lower	210 c c	Nil	30 8 33	Pleura free	Nil	No	No symptoms Gained 4 lb
29 C W	F 22	L	Lower	30 c c	Repeated	27 9 33	Pleura free Lobe adherent to dia- phragm	Nil	No	No symptoms ex- cept some lassitude on working
30 R A	F 17	L	Lower	60 c c Purulent	Occasional Free	11 10 33	Lower lobe ad- herent Upper free	Nil	No	No symptoms 21 3 34 Quite fit Gained 10 lb 18 4 34
31 H F	M 23	L	Lower	150 c c	Twice	11 11 33	Pleura free	Intercostal	No	No symptoms Gained 9 lb 21 3 34 (See Figs 206- 210)
32 H K	M 11	R	Lower	Scanty	Nil	15 11 33	Pleura free	Nil	No	No symptoms
33 H F	M 30	R	Lower	60 c c	Nil	22 11 33	Affected lobe very adherent Other free	Nil	No	Died 9 5 34 with residual empy- ema, T B of R lung, resulting in streptococcal effusion in R pleura and peri- cardium
34 S D	M 34 and L	R	Lower	60-90 c c	Severe quent	Fre- 27 11 33	Left lower lobe adherent per free	Nil	No	No symptoms Well and gained 7 lb 28 4 34 (See Figs 196- 199) Very fit Gained 11 1/2 stone appreciable spu- tum 9 5 34 (See Figs 200- 201)
35 J W	F 9	L	Lower	90 c c	60 c c	6 12 33	Mediastinal ad- hesions	Intercostal (1) Secondary (2)	Yes	
36 H R	M 38	R	Lower	50-60 c c	Nil	13 12 33	Generalized ad- hesions			

c c

LOBECTOMY FOR BRONCHITIS										
No.	Sex	Age	Side	Condition	History	Operation	Post-operative	Result	Remarks	No symptoms
37	M	23	L	Lower	900-1800 c c	Repeated	5 1 34	Generalized adhesions	Intercostal	No symptoms
38	J S	M 23	L	Middle and lower	90 c c foul	Once	17 1 34	Pleura free	Intercostal	Died 20 1 34 Suppurative bronchopneumonia
39	J K	M 46	R	Lower	60 c c green and offensive	Several	14 2 34	Pleura free	Intercostal	No symptoms
40	S T	F 47	L	Lower		Repeated	21 2 34	Pleura free	Intercostal	No symptoms
41	N M	F 43	L	Lower	A little	Repeated	22 3 34	All lobes densely adherent	Intercostal	No symptoms
42	M C	F 49	R	Lower	150 c c purulent pneumonia	Nil	11 4 34	Pleura free	Intercostal	Died Reactionary hemorrhage from raw area of chest-wall in hospital
43	K C	F 18	L	Lower	60 c c	Occasional Slight	11 4 34	Pleura free	Intercostal	Healed—convalescent
44	M F	F 22	L	Lower	60-90 c c	Frequent 300-600 c c about once a month	30 4 34	Pleura free Very firm adhesions over lower lobe	Intercostal	Still in hospital Small residual sinus, otherwise well
45	A L	M 35	R	Lower	30 c c	Two severe attacks	2 5 34	Lower densely adherent Upper partially adherent	Intercostal	Required retrograde removal of lobe Old drained abscess and bronchiectasis
46	L T	M 36	I	Lower	Small amount	Several attacks	4 5 34	Pleura free Lower lobe adherent to posteriorly diaphragm	Intercostal	Still in hospital Sinus healed Sinus valvulent in hospital
47	D H	F 22	L	Lower	210-240 c c	Repeated and severe	14 5 34	Pleura free except for fine band adhesions	Intercostal	Still in hospital—chest draining—condition good
48	C G	F 48	L	Lower	30 c c	Repeated and severe	14 5 34	Pleura free except for fine band adhesions	Intercostal	Still in hospital—chest draining—condition good

of hæmoptysis on several occasions, two of which were associated with the separation of silk ligatures, which were used for the suture of the hilum. In four others there has been a persistence of a certain amount of purulent expectoration, although this has been markedly reduced in every case. All these show evidence of some



FIG 196—Case 35. Skiagram showing atelectasis of left lower lobe partially obliterated by cardiac shadow.



FIG 197—Case 35. Bronchogram showing gross bronchiectasis of whole lower lobe. Note also outward curving of upper lobe bronchi.



FIG 198—Case 35. Bronchogram after lobectomy. Note stump of lower lobe bronchus and inward displacement of upper lobe bronchi.

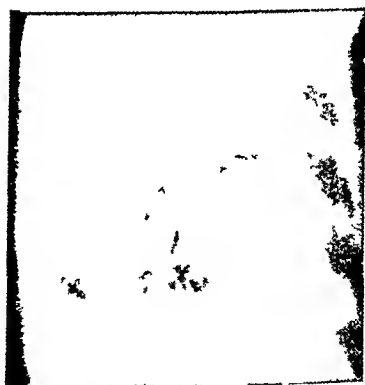


FIG 199—Case 35. Lateral bronchogram after lobectomy. Note complete filling of chest by expansion of upper lobe.

bronchiectasis in other lobes and would indicate a more careful investigation by lipiodol of the apparently unaffected portions of the lung. Thus it would appear advisable to carry out the investigation of each side independently, and particularly from the lateral view, if early stages of disease in the opposite side are to be eliminated with certainty.

The patient with the greatest quantity of purulent sputum following lobectomy now brings up about 60-90 c c daily, whereas before operation the quantity varied between 360 and 450 c c daily, and this has been shown to be due to bronchiectasis in the upper lobe of the same side. All these five are very definitely improved.



FIG 200—Case 36. Skiagram showing presence of portion of shell (old gunshot wound) and old fracture of seventh and eighth ribs.



FIG 201—Case 36. Lateral view showing shell fragment.



FIG 202—Case 36. Bronchogram showing cavitation around shell fragment and bronchiectasis of lower lobe.



FIG 203—Case 36. Lateral bronchogram showing cavitation.

The last of this group is a patient who has been symptomless for over three years and has recently developed tuberculosis of the opposite lung, which would appear to have no direct connection with the previous bronchiectasis and its eradication.

Twenty-nine patients are healed and symptomless, including one patient in whom malignant changes were found at the hilum of the bronchiectatic lobe.



FIG 204—*Case 36* Skiagram after lower lobectomy and late phrenicectomy

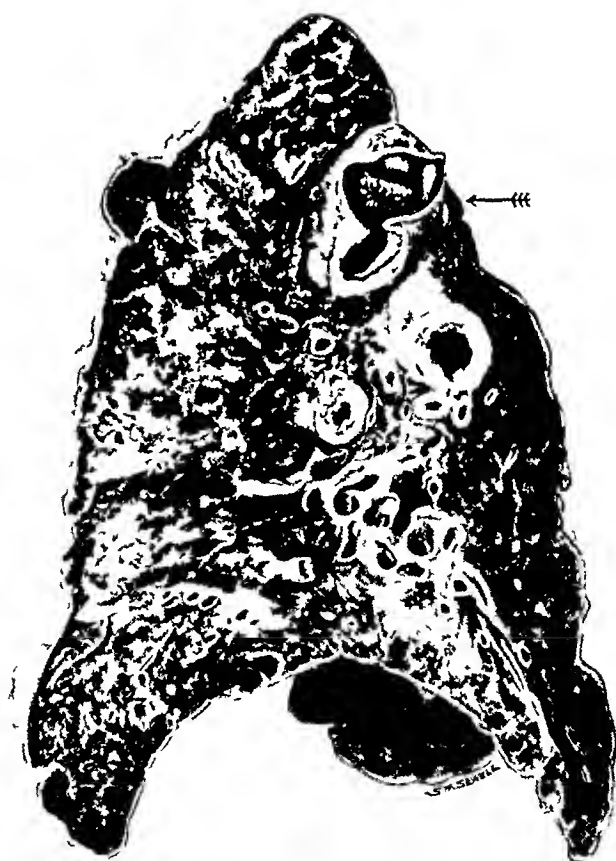


FIG 205—*Case 36* Gross generalized bronchiectasis secondary to cavitation due to the presence of a piece of shell from a gunshot wound during the War (the arrow indicates fragment of shell)

One other point might be considered here—namely, that in two patients the chest-wall adhesions were so dense laterally and posteriorly that the operation had to be modified. In both it was possible to free the area around the hilum where the adhesions were less firm and to thread the silk around the hilum, fix it in



FIG 206—Case 32. Skiagram showing atelectasis of right lower lobe



FIG 207—Case 32. Bronchogram showing area of bronchiectasis which comprises whole of lower lobe. Note the contraction in size and the relative expansion of upper lobe



FIG 208—Case 32. Lateral bronchogram showing bronchiectasis. Note both sides are filled with lipiodol and outline of normal bronchi below area of bronchiectasis corresponds to filling of unaffected left side



FIG 209—Case 32. Bronchogram after lobectomy

the metal part of the tourniquet, and, after tightening, to divide the pedicle. The lobe was now dissected off the chest-wall without excessive bleeding. Modification of the tourniquet after the first case permitted an easier operation in the second case.

Lastly, the operation here called one-stage lobectomy is meant to indicate that no attempt has been made to induce adhesions of the unaffected lobes to the chest-wall by a deliberate operation preliminary to the actual resection of the diseased lobe

SUMMARY

1 An account of 48 cases of the so-called one-stage lobectomy for bronchiectasis is given

2 Of these, 4 have died from conditions essentially associated with the operation itself, 3 have died later, 1 from cerebral abscess five weeks after operation, 2 from tuberculosis of the other lung, with terminal complications but each having a large residual empyema on the operated side, one three months and the other nearly six months after operation



FIG 210—Case 32 Lower lobe after removal. Note contraction of lobe which is solid and shows gross dilatation of the bronchi



FIG 211—Case 18 Gross generalized bronchiectasis in right lower lobe

- 3 Six patients, still in hospital, all show evidence of steady recovery *
- 4 Of the remaining 35, 6 have some residual symptoms of slight degree compared with their original condition, and 29 are virtually symptomless
- 5 This account does not include cases of lobectomy for malignant disease



FIG 212—Case 16 Large bronchiectatic cavity with generalized bronchiectasis of left lower lobe

except for one patient in whom bronchiectasis was the predominant lesion and an unsuspected carcinoma was disclosed at operation

6 Lobectomy, performed in one-stage, appears to be a justifiable procedure, and should be the operation of choice for unilobar bronchiectasis

BIBLIOGRAPHY

- POWELL, R D, and HARTLEY, P H-S *Diseases of the Lung*, 215
 JEX-BLAKE, A J, *Brit Med Jour*, 1920, May, 591
 GRAHAM, E, *Jour Thor Surg*, 1, 502
 LILIENTHAL, H, *Thoracic Surgery*, 11 Saunders & Co, 1925
 ALEXANDER, J, *Surg Gynecol and Obst*, 1933, March, 658
 BRUNN, H, *Ibid*, 1932, Nov, 616
 SHFNSTONE, N, and JAMES, M, *Canad Med Assoc Jour*, 1932, Aug, 138

*All 6 patients recorded in the above paper as "in hospital" have been discharged healed and symptomless. Nine more patients have undergone lobectomy for bronchiectasis subsequently with one death. Thus there is now a total of 57 patients, of whom 8 have died, giving a total mortality, early and late, of 14 per cent.

SARCOMA OF THE DUODENUM REPORT OF A CASE

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TUMOURS of the duodenum are rare. Primary tumours are said to be commoner than malignant,¹ and of the latter, carcinomata are more common than sarcomata. The relative incidence of the type of malignant tumour in the intestine may be judged from figures collected by Mueller,² who in 521 cases of carcinoma found 41 occurring in the intestine, and in 102 cases of sarcoma only 1 instance of involvement of the bowel. Similarly, Nothnagel³ found 243 cases of carcinoma of the intestine amongst 2125 carcinomata, 3 sarcomata of the bowel in 274 sarcomata, and 9 cases of lymphosarcoma of the bowel in 61 cases of lymphosarcoma.

Sarcomata may occur in any part of the bowel, but are more common in the small than the large intestine, where they are rare, with the exception of rectal ones, which are as common as those occurring in the small intestine. Libman,⁴ in collecting 52 cases where the sites had been mentioned, found 15 in the duodenum, 18 in the jejunum, 2 in jejunum and ileum, 14 in the ileum, and 3 in the entire intestinal tract.

References in the literature to sarcoma of the duodenum are scanty. In addition to the cases collected by Libman¹—which include all sarcomata of the small intestine, including the duodenum, cases have been described by Eiger,⁵ Shapiro,⁶ Sailer,⁷ Barber,⁸ Faykiss,⁹ Terentyeff,¹⁰ Mackenzie,¹¹ Angier and Fievez,¹² Bland-Sutton,¹³ Freud,¹⁴ Von Salis,¹⁵ Giani,¹⁶ Mostowska,¹⁷ David,¹⁸ Soli,¹⁹ Pfundt,²⁰ and Laroque and Shifflett.²¹ The majority of these cases have been published in journals to which, unfortunately, I have not been able to refer.

In view of the rarity of the condition it was thought to be of interest to put on record an additional case.

CASE REPORT

The patient, a married woman aged 51, was admitted to the Liverpool Royal Infirmary on June 9, 1933, complaining of epigastric pain and a swelling of the neck, each of two years' duration.

HISTORY—The pain in the epigastrium had begun two years ago, but had been negligible till two months ago, when it became more severe. The pain was sharp and stabbing in nature and radiated along the costal margin to the left axilla, but not to the back. It lasted about fifteen minutes at a time, and was unrelated to food. The patient had suffered from vomiting for the last three months, the vomiting occurring before breakfast usually, and occasionally during the day. Vomiting would relieve the pain—the vomit did not contain food, was 'yellow liquid'—the quantity being about a pint. There was a doubtful history of blood in the vomit on two occasions. For the past three weeks the patient had been free from this symptom. The appetite had been poor lately. The bowels were regular. She suffered considerably from flatulence between meals. She had lost 22 lb. in weight in the last nine months. There was no urinary disturbance.

Apart from the digestive symptoms, the patient had noticed a swelling in her neck for the past two years, which, she thought, fluctuated in size. She also had been suffering from

nervousness and palpitations for the past six months. There was no history of dysphonia or dysphagia.

ON EXAMINATION—She was a small, thin, pale woman and had evidently lost weight. There was no gross anaemia.

Thyroid—There was slight enlargement of both lateral lobes, and an adenoma was present in the isthmus. The latter was $1\frac{1}{2}$ in diameter, smooth, soft, fluctuating, and situated in the middle line. There were no eye signs of thyrotoxicosis nor tremors of the fingers.

Abdomen—A small mass 2×1 in was palpable in the right hypochondrium, it was not very hard, slightly tender, and one could get above it. The swelling was slightly movable, though it did not move on respiration. The swelling did not pass into the loin. It was dull to percussion. There was nothing else palpable in the abdomen. Rectal examination revealed no abnormality.

Cardiovascular System—The pulse was 100 on admission. The blood-pressure was 210 mm systolic and 170 diastolic. The heart was slightly enlarged, there was a systolic murmur at the mitral area with reduplication of the second sound there.

The respiratory and central nervous systems showed no gross abnormality.

Investigations—Radiological examination of the gastro-intestinal tract showed gastroptosis with no delay and no evidence of organic lesion of the stomach or duodenum. There was some colonic stasis. A barium enema, in confirmation, revealed no colonic lesion.

Examination of the gall-bladder by Graham's method revealed a radiologically normal gall-bladder. X-ray examination of the neck showed no tracheal compression.

TREATMENT—The patient was thought to be suffering from a toxic adenoma of the thyroid—apart from her abdominal condition—and was treated with Lugol's iodine 5 min three times a day, and luminal $\frac{1}{2}$ gr three times a day, during the time she was being investigated.

In view of the presence of the tumour, it was decided to explore the abdomen. This was done on July 10 by Professor R. E. Kelly, under whose care the patient was. Under intratracheal anaesthesia administered by Dr. Bennett Jones, the abdomen was opened through a right paramedian incision. A swelling could be felt on the posterior abdominal wall. The stomach, omentum, and transverse colon were turned up and a tumour was made out in the neighbourhood of the third or transverse part of the duodenum, just beyond the centre.

On incising the peritoneum, the swelling was found to be partly within the duodenum and partly without it, though it seemed definitely to be arising from it. It was situated on the anterior wall and nearer the superior border, so that the extra-mural portion—the bigger—was within the hollow of the curve formed by the duodenum. There was no dilatation of the proximal duodenum, but there was some slight hypertrophy of its wall. The swelling with a cuff of duodenum round it was resected, and the hole in the duodenum sutured transversely so as to obviate narrowing of the gut.

The superior mesenteric artery was identified and held aside during the procedure. It was felt that the suture was not altogether safe, and to relieve any possible tension, a posterior gastro-enterostomy was performed. The peritoneum over the posterior abdominal wall was sutured, and the abdomen was closed without drainage.

POST-OPERATIVE COURSE—The patient's pulse rose to between 120 and 130, her temperature being about 100° on the third day following the operation, the pulse rose still higher to 140, the temperature at the same time rising to 103.4° . She died on the following day, with a temperature of 105° and a pulse varying from 150 to 160.

AUTOPSY—A limited examination through the incision was made after death. The abdominal cavity was normal, there had been no leaking, the sutures had held, and the anastomosis was sound. There was no distension of the stomach or bowel. Death was probably due to post-operative exacerbation of thyrotoxicosis.

Gissel,²² in discussing thyrotoxicosis, mentioned two deaths, one following operation for carcinoma mammae and the other a hernia, in patients suffering from thyrotoxicosis. He points out the importance in these cases of pre- and post-operative medication for the thyroid condition, similar to that given them prior to subtotal thyroidectomy. Both his cases had had pre-operative iodine, but in the one iodine had not been given after operation, and in the other it had been stopped owing to the onset of influenza. The patient here reported had pre-operative medication, but no post-operative iodine, except prior to death.

EXAMINATION OF SPECIMEN (Fig 213)—This resembled in form a uterus, the part protruding within the duodenum being very like the vaginal cervix, the part outside like the fundus and body of the uterus, whilst the intervening portion was embraced by the duodenal wall. After fixation the dimensions of the tumour were approximately 5.4 cm in length, the upper end was 4.6 cm \times 3.2 cm in area, whilst the intraduodenal part was



FIG 213 —Microscopic appearance of tumour. A, Extramural part, B, Duodenal cuff, C, Intraduodenal part

2.2 cm in its broader and 1.7 cm in its narrower diameter. The length of tumour outside the wall was roughly 2.7 cm, the part within the lumen 2.3 cm, and the remaining 0.4 cm was intramural.

The intraduodenal portion was smooth, not ulcerated, the free end having a depression in it about 0.2 cm deep. The outer portion was smooth, but close to the wall of the duodenum, arising from the main mass, there was a small pedunculated tumour, rather flat, and very like a tiny subperitoneal fibroid. The tumour was fairly hard, and dull fleshy in colour.

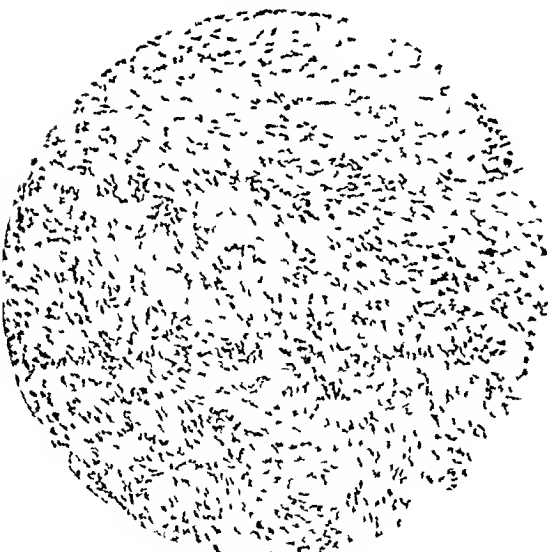


FIG 214 —Microscopic appearance of tumour (\times 50)

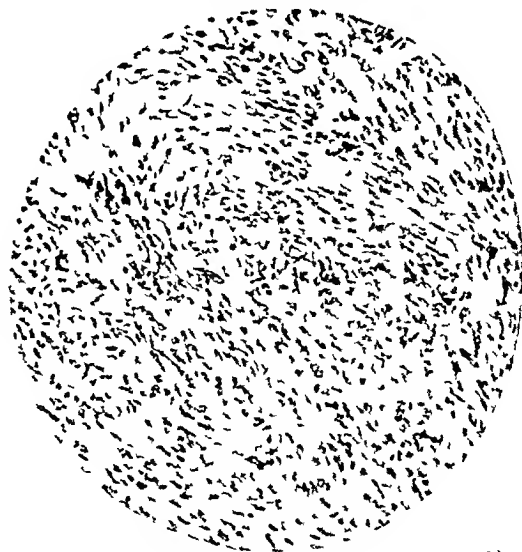


FIG 215 —Microscopic appearance of tumour (\times 105)

A microscopical examination of a piece taken from the 'fundus' was made by Professor J. Henry Dible, who reported: "This is a borderline growth of unstriped muscle—myosarcoma. I think it is the sort of case in which recurrence may be looked for" (Figs 214, 215).

DISCUSSION

Etiology —

Age—Libman, collecting 51 cases of sarcoma of the small intestine where the age was noted, found the distribution to be as follows First decade, 6, second, 9, third, 13, fourth, 13, fifth, 8, sixth, 1, and seventh, 1

Sex—In the cases collected by Libman the condition was found to be two and a half times as common in men as in women

The causation is unknown A case has been described (Bland-Sutton¹³) in which the sarcoma, spindle-celled in type and situated in the first part of the duodenum, was associated with the scar of a healed duodenal ulcer The association—in view of the frequency of duodenal ulcer and rarity of sarcoma—was almost certainly fortuitous

Pathology—All varieties of sarcoma have been described, mixed-celled sarcoma, lymphosarcoma, spindle-celled sarcoma, myosarcoma, endothelioma, malignant melanotic and round-celled sarcoma, though the latter perhaps should be classed with the lymphosarcoma Secondaries occur especially in lymphosarcoma, possibly the commonest type found in the small intestine

The tumour is usually primary in the duodenum, though secondary tumours are also described—for example, the case of lymphosarcoma arising in the retroperitoneal glands and secondarily involving the duodenum described by Moore,²³ and the case of spindle-celled sarcoma of the first part of the duodenum described by Laroque and Shifflett²¹—the primary being a fibrosarcoma of the thigh

Symptoms and Clinical Course—The length of history is usually short—two weeks to twenty-one months (Baltzer²⁴) Rarely, however, the history is of longer duration—for example, in the case of myosarcoma of the duodenum reported by Von Salis, where the history was of seven years' duration¹⁵

The symptoms are those of dyspepsia—perhaps the pain may be colicky There is usually vomiting, loss of appetite, and loss of weight, occasionally there is a history of blood appearing in the vomit The tumour may further give rise to symptoms because of (1) Pressure on surrounding structures (*a*) the vena cava, causing œdema of the legs and ascites, (*b*) the bile-ducts and pancreatic duct (Lancereaux,²⁵ sarcoma of the duodenum), (*c*) the ureters, (2) Ulceration—which may lay open vessels—for example, Rolleston's²⁶ case of sarcoma of the duodenum (probably a lymphosarcoma) which eroded the inferior pancreatico-duodenal artery with consequent fatal hæmatemesis Palpable tumours are uncommon Where metastases occur, these are more likely to be a factor in the diagnosis The characteristics of the tumour are those found in the case recorded

The history is not suggestive, nor are the physical signs, and these cases were at one time diagnosed either at laparotomy or post-mortem Latterly, however, diagnoses have been made based on radiological findings Freud¹⁴ thus was able to diagnose a sarcoma of the fourth part of the duodenum and upper part of the jejunum with metastatic deposits in the bowel At operation multiple metastases were found on the bowel with infiltration of the walls of the terminal duodenum and some dilatation

The tumour gives rise to a vacuolar filling defect, and if this is associated with dilatation of the duodenum proximal to it, a diagnosis of tumour of the duodenum

may be made. The defect is best shown with the patient in the right oblique position (Waters)²⁷

The tumour in the case recorded was in the transverse part of the duodenum and unassociated with dilatation or stenosis, so that its demonstration by radiography must be very difficult

Treatment—This is surgical. The majority of tumours are situated in the first and second part of the duodenum, so that this may be excised and an end-to-end anastomosis with the stomach performed. In the third part a local excision is probably the best operation with a short-circuit to relieve tension from sutures placed in a viscus only partly peritonealized

SUMMARY

- 1 A case of myosarcoma of the duodenum is reported
- 2 The difficulties of diagnosis are noted
- 3 The necessity for pre- and post-operative medication in cases of thyrotoxicosis, whatever the operation may be, is stressed

It is a pleasure to express my thanks to Professor R. E. Kelly, under whose care the patient was and who performed the operation, for permission to publish this case. I am indebted to Dr R. E. Roberts for permission to publish the radiological findings, and to Professor J. Henry Dible for the microscopical report on the tumour

REFERENCES

- ¹ KELLOGG, E. L. and W. A., *Amer Jour Surg*, LV, 268
- ² MUELLER, Quoted by Libman
- ³ NOTHNAGEL, Quoted by Libman
- ⁴ LIBMAN, E., *Amer Jour Med Sci*, 1900, CV, 309
- ⁵ EIGER, I. B., *Bolnitsch gaz Botkina*, St Petersburg, 1895, VI, 921, 950
- ⁶ SHAPIRO, G. A., *Vrach St Petersb*, 1897, VIII, 1205, 1235
- ⁷ SAILER, J., *Trans Pathol Soc Phila*, 1898, VIII, 122
- ⁸ BARBER, W. L., *Proc Connect Med Soc*, 1901, 260
- ⁹ FAYKISS, F., *Budapesti Kmt cgyet 2 sz Seb Klin betegf*, 1903-4, Budapest, 1905, 49
- ¹⁰ TERENTYEFF, A. F., *Voycimo-Med Jour St Petersb*, 1906, CCVII, Med Spec pt 16-26
- ¹¹ MACKENZIE, H. M., *Lancet*, 1909, I, 1823
- ¹² ANGIER, D., and FIEVEZ, J., *Jour de Soc med de Lille*, 1912, I, 433
- ¹³ BLAND-SUTTON, Sir JOHN, *Lancet*, 1914, II, 931
- ¹⁴ FREUD, J., *Wien Illn Woch*, 1916, XXIV, 1460
- ¹⁵ VON SALIS, H. W., *Deut Zeits f Chir*, 1920, CLX, 180 (Abstr Jour Amer Med Assoc, 1921, LXVI, 972)
- ¹⁶ GIANI, E., *Arch di pat e clin Med*, Bologna, 1924, III, 9
- ¹⁷ MOSTOWSKA, Z., *Polsha gaz lek*, 1927, VI, 250
- ¹⁸ DAVID, V., *Casop lek cesk*, 1929, LXVIII, 1607, 1648
- ¹⁹ SOLI, D., *Clin Chir*, 1930, XXXIII, 887
- ²⁰ PFUNDT, W., *Arch f klin Chir*, 1931, CLXIII, 488
- ²¹ LAROQUE, G. P., and SHIFLETT, E. L., *Ann of Surg*, 1933, LXVIII, 178
- ²² GISSEL, H., *Trans Pathol Soc Lond*, 1882-3, XXIV, 99, *Brit Med Jour Epitome*, 1933, Dec, 98
- ²³ MOORE, N., Quoted by Libman
- ²⁴ BALTZER, Quoted by Libman
- ²⁵ LANCERAUX, H. D., *Trans Pathol Soc Lond*, 1892-3, LIII, 67
- ²⁶ ROLLESTON, H. D., *Trans Pathol Soc Lond*, 1892-3, LIII, 67
- ²⁷ WATERS, *Amer Jour of Rontgenol*, 1930, XXIV, 554 Quoted by Laroque and Shiflett

THE ESTABLISHMENT OF A LARYNGEAL BY-PASS

By H P PICKERILL, CBE, SYDNEY

THIS note is the record of an attempt to avoid the disadvantages of a permanent tracheotomy tube, and at the same time conserve a sufficient airway without impairment of the voice

The patient, a good-looking, petite girl, aged 25, had a partial thyroidectomy performed in January, 1930. This was followed by increasing dyspnoea for eighteen months, with an extremely weak but distinct voice. In August, 1931, a tracheotomy was done and the tube worn constantly until I saw her in December, 1932. Dr. Garnet Halloran (laryngologist) then reported: "Examination of the glottis reveals bilateral abductor paralysis. There is only the slightest movement of both cords on phonation or inspiration. The resultant glottic chink would not be sufficient without a tracheotomy tube."

Various laryngeal operations were discussed—decortication of the cords, and so on—all of which the patient declined on being told that they would undoubtedly affect her voice adversely. After considerable thought, therefore, and having explained the details to the patient, I determined to endeavour to establish a by-pass airway, as I concluded that anatomically, if not surgically, this was possible. The objective was to establish an additional airway which would pass external to the laryngeal cartilages but underneath the skin, from the tracheotomy opening in the trachea to a point in the larynx above the constricted vocal cords. I formed the opinion that there was a small area between the cushion of the epiglottis and the false vocal cords which might be utilized for the insertion of a tube, and at which point air would be free to pass on inspiration and expiration, but which would be closed on deglutition. I fully explained all these things to the patient, who, I must say, entered into the plan of campaign with considerable interest and understanding. "Anything," she said, "to get rid of this horrible hole in my neck, so that I can surf bathe and dance like other girls."

My plan was first of all to establish a skin-lined subcutaneous tube passing from the tracheotomy opening to an opening in the larynx made just at the notch in the upper border of the thyroid cartilage. I had a solid core of vulcanite made of correct size and shape, and then applied a Thiersch graft to it, and inserted it subcutaneously. Owing, however, to the smallness of the patient's neck and the comparative tightness of the skin, this could not be inserted satisfactorily and would not stay in place. I therefore fashioned a larger silver tube, made in two parts and connected in the middle by a sliding tube (*Fig 216*). The lower half of the silver tube was inserted into the tracheotomy opening and turned so as to resemble, and function as, an ordinary tracheotomy tube (*Fig 217*). An incision was then made over the upper part of the thyroid cartilage and thyro-hyoid space. The cartilage of the thyroid notch was then deepened a little. Next, with the direct laryngoscope held in place by the anaesthetist, I was enabled to pass a sharp-pointed

probe from the outside to the inside of the larynx at the exact spot at which I wished it to be—namely, just above the false vocal cords and below the cushion of the epiglottis. With the probe in position a bistoury was passed alongside it, and a vertical incision made through the thyro-hyoid membrane and mucous membrane of the larynx. This was dilated and the flange-like end of the upper silver tube insinuated into place and observed via the laryngoscope to be lying snugly in position. There was no trouble from any hæmorrhage into the larynx.

Whilst the patient was still under intratracheal anæsthesia, the by-pass was tested by joining the two ends with the sliding tube, and apparently it functioned well. In fact there was an obvious improvement in respiration. But would the patient be able to swallow without fluids entering the tube, and therefore the trachea? Here, of course, was the object of the two-part tube. The two portions

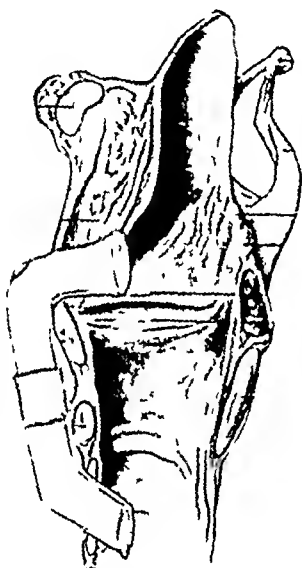


FIG. 216 — Semidiagrammatic view of larynx with by-pass tube in position. The two parts of the tube united in the middle by a sleeve.



FIG. 217 — Semidiagrammatic view of the larynx showing by-pass tube in two parts in position—the lower half turned to act as a tracheotomy tube, and the upper half left open for observation.

were left in position unconnected, but with the lower end turned to the tracheotomy position until the day after the operation, as shown in *Fig. 217*. The patient was then given a little water to drink and we were very gratified to find that none came through the lower end of the upper part of the tube. The two tubes were left thus for several days, until swelling and tenderness had subsided, and it was obvious that deglutition could take place without any danger of either fluid or food particles entering the upper end of the tube, and then the two tubes were connected in the middle by the sliding tube as in *Fig. 216*. From that point we progressed slowly, but with complete success, to normal feeding, and connecting up the two parts of the tube. We had demonstrated that a laryngeal by-pass was physiologically possible. Respiration was easy and comfortable, the voice definitely stronger, and deglutition normal.

The by-pass tube was during this time lying external to the skin, held in place

by strapping and tape. To cover the silver tube I fashioned a Thiersch-lined tube-graft from the chest, and swung the lower end up to a prepared bed under and around the exposed portion of the silver tube. Thus the tube was not visible, though the graft and the subjacent tube made a prominence on the neck something like an exaggerated 'Adam's apple'. All this occupied several months. When the patient was last seen, the apparatus was functioning excellently. Voice, respiration, and deglutition were normal. The patient could bathe, she danced again after three or four years, and she has been in the cold country amongst snow without any untoward effect. The only disadvantage was the difficulty in keeping the tube clean. It was found that a film of mucus collected and this had a disagreeable odour. To overcome this I exposed the upper bend of the tube, drilled a hole in it, and had a small silver screw stud fitted. Thus the patient removes and cleans the tube with pipe-cleaners dipped in hydrogen peroxide. The stud she covers with a piece of jewellery or a narrow silk scarf.

In the original scheme of things it was proposed to remove the silver tube later, and graft in cartilage rings to maintain patency, but this awaits the patient's decision. In the meantime it is of sufficient interest to record that a laryngeal by-pass is anatomically, physiologically, and surgically possible.

I may say that the success of this somewhat tedious series of operations was largely due to the excellent assistance and suggestions of Dr C M Clarkson (assistant) and Dr Campbell Lamrock (anæsthetist).

EXCISION OF THE ŒSOPHAGUS FOR CARCINOMA

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HAVING read of Mr Grey Turner's successful case and studied his Bigelow Lecture¹ advocating the pull-through method which he had evolved, I was very much struck by the avenue of hope laid open in an almost hopeless disease. About this time a man of 52 years presented himself with a five months' history of difficulty in swallowing. An œsophagoscopy was performed and a growth seen high up in the thoracic œsophagus (*Fig 218*). A piece of the growth was taken for biopsy and the

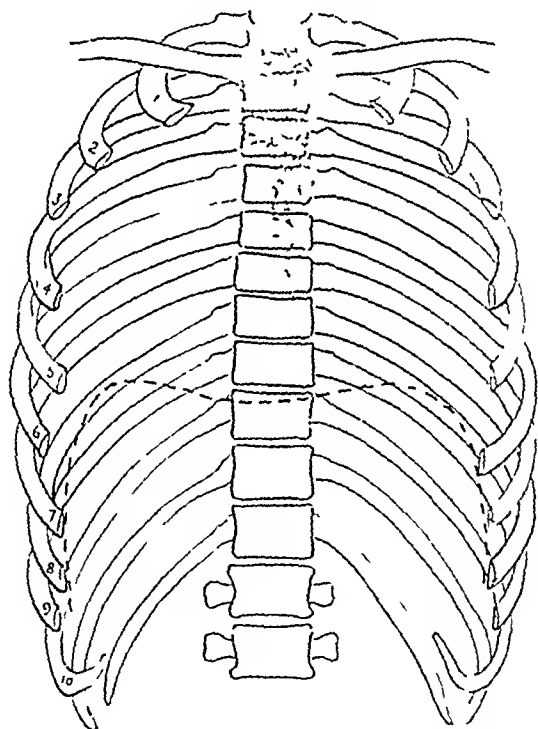


FIG 218—Drawing from a skiagram showing position of the growth

diagnosis of carcinoma was confirmed. At the same time a laryngoscopic examination was done and the left vocal cord was seen to be immobile, gastrostomy was carried out the following day. The patient was in fairly good condition, weighing 8 stone on admission, and 8 stone 2 lb three weeks later, when the œsophagectomy was performed following a blood transfusion.

OPERATION—The intratracheal method of anæsthesia was adopted, giving gas, oxygen, and ether. The gastrostomy was excluded from the field of operation by a temporary collodion seal and the Grey Turner technique followed—i.e., a

vertical incision from the apex of the left costoxiphoid angle to the umbilicus, the mobilizing of the left lobe of the liver by cutting through the left lateral ligament by means of a pair of angled hysterectomy scissors and thus approaching the lower end of the œsophagus. It was found that by using the left hand as a retractor the left lobe of the liver was kept out of the way without withdrawing it from the abdomen, whilst the hand of an assistant kept the stomach out of the way on the opposite side. By this means a narrow lane was formed, at the end of which the terminal œsophagus could be plainly seen, and about 20 c c of 2 per cent novocain were injected beneath the peritoneum into the tissues surrounding the lower end of the œsophagus. A small transverse nick about $\frac{1}{4}$ in long was made through the peritoneum covering the terminal œsophagus and the tip of the right index finger insinuated into the subperitoneal tissues and the enucleation commenced. It was not a difficult manœuvre, and I think perhaps the secret lies in working in the tissues deep to the nerve plexuses, and one had the sensation of working inside a string tube, as long as one kept inside the strings the separation was easy. It was estimated that the maximum height reached by the abdominal finger was the second intercostal space (anteriorly). The abdominal wound was protected by towels and the cervical œsophagus exposed by a transverse incision dividing the left sterno-cleido-mastoid muscle completely and the œsophagus identified and mobilized.

As the downward enucleation proceeded it was found that at about the level of the second dorsal vertebra there was a dense mass firmly adherent to the dorsal vertebra behind and to the trachea in front (*Fig 219*). These were broken down by digital pressure as far down as possible, but not far enough to reach the upward dissection. At this stage the cervical œsophagus was divided and the cut ends were 'bipped'. By virtue of firm traction from below and enucleation from above, the œsophagus was pulled downwards, divided near the stomach, and the stump buried by means of a purse string. It was then noticed that the stomach was suspended by its vagus nerves and that the left lobe of the liver when released resumed its normal position, a finger was passed to the site of the peritoneal incision to estimate its size and it was difficult to find because of its smallness. The skin incisions were closed, taking care in the neck incision to suture the medial edge of the sterno-cleido-mastoid muscle to the prevertebral fascia. The cervical œsophagus was stitched in the medial corner of the incision.

SUBSEQUENT HISTORY—The patient did not seem to be upset by the operation, he had a pulse-rate of 90 and there was very little bleeding. After the separation of the growth from the posterior aspect of the trachea it was noticed that he coughed up some blood-stained sputum. At no stage during the operation did his condition give rise to any anxiety.

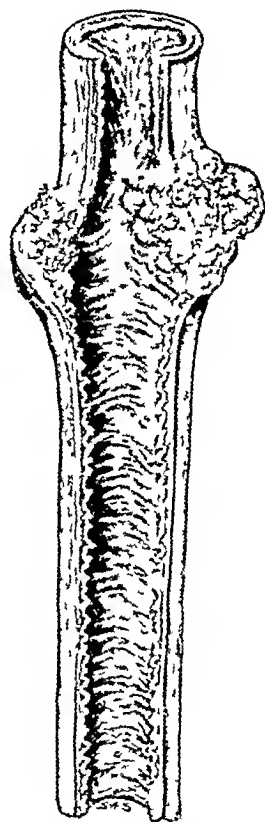


FIG 219 —The œsophagus has been slit up on its ventral aspect and shows that part of the growth adherent to the trachea

The following day the sputum was rusty, but his condition was quite good. The next day he had difficulty in breathing, the temperature was raised, and he unfortunately died.

AUTOPSY—At a post-mortem it was found that the trachea was infiltrated by growth and had been injured in separating the dense adhesions, no macroscopic breach in continuity of the tracheal mucous membrane could be detected, but the fact that he had blood-stained sputum coupled with the microscopic report on the trachea involved pointed the cause of the failure.

COMMENTARY

The points that are instructive in this case are (1) The paralysis of the left vocal cord, (2) The involvement of the trachea. With regard to the first point, and in the light of the findings at operation, the left recurrent laryngeal nerve was manifestly involved, and by comparison with facial palsy complicating malignant parotid tumours, one may safely assume that this was an actual involvement by growth. Touching the second point, it is felt that the pre-operative investigations might have been rounded off by an endoscopic examination of the trachea and/or bronchi, and that any undue inflammation of the mucosa should preclude operative interference.

To sum up, it would seem that operation is not contra-indicated by a laryngeal palsy provided the surgeon is prepared to bury radon seeds in the infiltrating growth not removed, on the other hand the operation is contra-indicated when it can be established that involvement of the trachea or a bronchus is present.

The lessons learnt from the actual operation were (1) The absence of shock to the patient, (2) The absence of severe hæmorrhage, (3) The tendency for the left lobe of the liver to resume its normal position without suspension to the diaphragm by suture, (4) That a small initial peritoneal incision relieves one of the necessity of stitching the liver over to prevent herniation and that it is possibly an advantage to have a lower outlet to the traumatized œsophageal tunnel, (5) Provided one enucleates inside the nerve plexuses and leaves the vagi intact there is a natural suspender for the cardiac end of the stomach.

REFERENCE

- TURNER, G. G., "Some Experiences in the Surgery of the Œsophagus", *New Eng Jour Med*, 1931, ccc, 657.

VISITS TO SURGICAL CLINICS AT HOME AND ABROAD

THE ACCIDENT HOSPITAL OF DR BOHLER IN VIENNA

THE origin, structure, and organization of this hospital are all so unusual that it is necessary to devote a few introductory words to them before describing the incidents of a recent visit

In modern days it is the fashion to make hospitals large, expensive, and showy, but Dr Bohler has been quite independent of any great outlay of money. He saw and seized a unique opportunity of utilizing a building designed for another purpose, and during the last nine years he has formed a wonderful hospital organization, which, instead of costing money, has paid for itself and its staff and saved money for the community

Dr Bohler's experience of traumatic surgery, especially in the treatment of fractures, during the War, made him realize the terrible waste of lives, of limbs, and of working capacity that results from the neglect of prompt, efficient, and thorough treatment of wounds and other injuries. Therefore in 1919, when the end of the War left him free to turn his attention to peace problems, he offered his services to his own country, Austria, in order to save the victims of industrial accidents from mutilation, invalidity, and incapacity. In 1912 there had been built on the south bank of the Danube, in Vienna, a large building, six stories high, for the offices of the National Insurance Company. This was designed on a large scale, so as to be able to deal with the working population of the Austro-Hungarian Empire, which included 56,000,000 people. After the re-distribution of Central Europe this Company had only to insure the working people of Austria, with its population of 6,000,000. The building was therefore out of all proportion to the needs of the office. Dr Bohler suggested that part of it should be converted into a hospital for the treatment of insured persons. His idea was accepted in principle, but just at that time the financial crisis caused by inflation was so severe that all new developments were suspended, and it was not until 1925 that the suggestion was carried out (*Fig 220*)

The upper two floors of the insurance building were then adapted as a hospital, accommodating 120 in-patients, the operating- and treatment rooms, X-ray room, and offices, as well as affording residence for the nursing and surgical staff. The wards, thirty-two in number, are arranged on the outside of two sides of a square, whilst the kitchens and offices lie on the corresponding inner sides. The largest wards contain five beds, the smallest three, and, although lofty and well ventilated, are rather overcrowded as compared with our modern hospital standards, but not so much crowded as is usual in continental hospitals, such as the General Hospital in Vienna.

The visitor approaches the hospital, then, through the entrance of the insurance company's offices. As he takes the lift to the fourth floor, or climbs the one hundred stairs, he realizes that the hospital is only a department of the insurance business. On the landing of the fourth floor there are two doors, one leading into the hospital and the other into Dr Bohler's private residence. The main examination and treatment rooms are six in number, arranged in a series opening into one another, or rather communicating with a common passage. An office where records are stored and X-rays are examined, and the X-ray room, with couches for two patients, are at one end of this series. The X-ray plant, which includes



FIG. 220.—The 'Unfallkrankenhaus'

a very efficient portable machine, is remarkable for its simplicity. Every year between 12,000 and 13,000 pictures are taken, the majority by one of the nursing sisters, the others by one of the assistant surgeons. Both the X-ray apparatus and the films themselves are available for use or reference at any time by day or night. Next to the X-ray room is a receiving-room where minor surgery is done and where the examinations and preliminary treatment of all new out-patient cases are carried out. This can be divided by curtains into several parts, so that three or four cases are usually being dealt with at the same time. Then come two operating-rooms, separated from one another by a room for instruments, dressings, and sterilization.

The surgical staff (*Fig 221*) consists of Dr Bohler, two chief assistants, four secondary assistants, and two junior assistant surgeons. One of the chief assistants lives outside the hospital and is allowed to do private practice, the others are residents and give all their time to the hospital. The whole surgical staff are at work every week-day from 7.30 a.m. to 2 p.m., and for the rest of the twenty-four hours and on Sundays there are always four surgeons on duty, day and night. The first assistant has been with Dr Bohler since the opening of the institute nine years ago, the others for two or three years, thus affording an eloquent testimony to the enthusiasm which he inspires.

The nursing staff is twenty-four in number, including a matron and six sisters. They have been trained in one of the training schools of Vienna.

On entering the treatment department one is at first confused by the many things which are going on at the same time—an almost bewildering activity—two



FIG 221—Dr Bohler and his assistants

sets of X-rays being taken, others being developed and inspected, a recent case having the wound excised, a fractured arm or leg being reduced under local anaesthesia and put up with pin or wire traction and the application of plaster, a major operation such as the pegging of the neck of a femur in one theatre, and the setting of a fractured tibia by screw traction, transfixation, and plaster in the other. But it soon becomes apparent that this manifold activity is not confusion, but merely the organized activity of the beehive where every unit has his appointed task, which he does without interfering with the others.

Over two-thirds of the patients are working people, insured against accidents. The remainder consist of private patients, six to twelve in number, and those cases of accident too urgent for inquiry to be made before admission. The private patients pay 20 to 25 shillings* a day, and the others 8 to 11 shillings. All insured

* The shilling referred to in this article is the Austrian schilling of which 27 go to £1 sterling.

poisons have a right of admission to this hospital, but there is no compulsion. In 1932 there were 1916 in-patients, with an average stay in hospital of seventeen days, and 6478 out-patients, with an average period of fifteen days. The total cost of treatment during this year (1932) was £15,300, and it is estimated, by a careful comparison with the same number of cases treated elsewhere in unorganized clinics, that a sum of money is saved which is just double the cost of treatment. This is no rough-and-ready estimate but is very amply borne out by a great wealth of statistical information. It is thus made perfectly clear that this hospital organization is a sound business proposition, not dependent on sentiment, charity, or mendicancy.

The major operations are done three days a week, from 7.30 to 10.30, after which Dr. Bohler does a round of the wards accompanied by all the assistants who are not engaged in the receiving-room, a sister and nurses responsible for each ward, and visitors. On each round about forty cases are seen and examined in detail, the rest cursorily. Every patient is examined and a note taken once a week. An X-ray-viewing apparatus is available in every ward, and the X-rays are seen of all new patients and those patients examined in detail at the weekly visit.

But the examination and report of the X-rays is much more formal than this. On the three mornings a week on which there are no major operations, at 9 o'clock Dr. Bohler and all his assistants sit down to a meticulous examination and report of all the X-rays taken during the past few days. The frontal and lateral films are put up in the viewing-box, the history of the case is given in brief, and one of the assistants is invited to describe it. A discussion of the nature of the injury follows, and then Dr. Bohler dictates his own notes and decides on the treatment or modification of treatment which is to be adopted. He is now engaged in an intensive study of fracture-dislocations of the cervical spine, and when the films of a new case of this injury were presented, antero-posterior, lateral, and oblique, they were examined in very great detail, the case proved to be a rotatory dislocation on the left side, between the 3rd and 4th vertebræ, with the fracture of the tip of the 4th articular process. The articulated vertebræ from a skeleton were at hand, and the exact displacement was demonstrated by their aid. The trivial fractures and dislocations of the bones of the wrist, fingers, and toes were all noted and described with equal care.

It will make for simplicity if we first describe the operations we saw and then some of the patients in the ward, to illustrate the chief points in Bohler's principles and practice.

OPERATIONS WITNESSED

1. Open Fracture of Finger.—This was one of the little finger in a man of 69. The finger had been torn on the dorsal surface, from above the knuckle to below the first interphalangeal joint. Local anæsthesia was used, the skin was not washed but painted with iodine, the torn skin edges were excised, leaving a considerable open wound. The proximal phalanx had a T fracture into the joint. The defect of the skin was made good by a free skin-graft from the thigh. The finger was fixed on a suitable bent-wire padded splint and the whole arm was elevated on an aeroplane splint, the patient being admitted to the ward. He had been advised to have the finger amputated, but he begged that it might be saved. We saw him several days later in the ward, too soon to say whether

the skin-graft would survive (We have heard subsequently that the skin-flap did survive) The great care taken over this comparatively trivial injury was very impressive, and we found it characteristic of the whole work

2 Open Operation for Ununited Fracture of the Radius.—A man of 30 with fracture of the radius and ulna (November, 1933) The ulna had united but the radius presented a pseudarthrosis It had been treated by double transfixion (olecranon and metacarpals), traction, and plaster-of-Paris Later both ends of the bone had been subjected to multiple subcutaneous drilling by a wire drill 0.5 mm in diameter It was intended to do this operation under brachial plexus anaesthesia, but for some reason it was impossible to locate the brachial plexus with the needle, and anaesthesia was by open ether Through an incision about six inches long the radius was exposed at the site of fracture The pronator teres lay obliquely between the fragments and had obviously been responsible for non-union The interposing muscle was cut away, and both bone-ends were cleared



FIG 222 —Excision of wound of the finger under plexus anaesthesia

of scar tissue and refreshed by a nibbling forceps, until the marrow cavity was exposed in each The oblique fracture could then be held in good position by Lambotte's forceps The fragments were united by a single strand of stainless iron wire, which perforated and surrounded the bone All the scraps of bone which had been removed were packed round the area of fracture The wound was closed by three layers of interrupted sutures The patient was removed to the adjoining room and the arm put up in an unpadded plaster cast, extending from the shoulder to the knuckles, the elbow at a right angle and the forearm midway between pronation and supination The whole limb was finally placed on an aeroplane splint

3 Removal of Chondroma from the Knee-joint—A man of middle age who had had intermittent pain and disability in the left knee A tentative diagnosis of loose body in the joint had been made, the X-rays being negative Under spinal

anæsthesia the knee was opened by a longitudinal incision, medial to the patella. A fibro-cartilaginous mass was found growing from or attached to the deep surface of the vastus medialis, it was removed. The knee was closed by two layers of interrupted silk suture.

4 Dislocation of the Shoulder.—A stout woman of 55, who had had an accident three weeks previously, she had been treated elsewhere, but on account of the great swelling of the shoulder the nature of the injury had not been recognized. There was an anterior dislocation of the shoulder, the great tuberosity being fractured but not separated. Under general anæsthesia by ethyl chloride the shoulder was reduced by steady manual traction, counter-traction being made by the heel in the axilla. After reduction was effected, the arm could easily be put in and out of its socket. It was bandaged to the side. X-rays showed reduction to be satisfactory. In a week's time it would be put up on an aeroplane splint.

5 Old Fracture of the Neck of the Femur.—Johansen's modification of Smith-Peterson's method. A healthy man of 50 had had a fracture of the neck of the femur in February, 1934, and had been treated elsewhere by pin



FIG. 223.—Case of gunshot fractures of the femur occurring in the rioting in 1 February, 1934.
a, On admission, b, Three months later.

traction and plaster casts. He presented a typical medial fracture of the femoral neck, with shortening and varus deformity. Three days previously a pin had been put through the tibial crest and weight traction applied. X-rays showed the reduction was incomplete. Operation took place under spinal anæsthesia on June 8, 1934. Powerful traction was made by a screw traction apparatus. X-rays taken by a portable apparatus showed reduction to be satisfactory. Three small pieces of metal were stuck on to the skin along the line of the neck of the femur for purposes of orientation. Two long boring wires were then thrust about 11 cm. from the base of the great trochanter upwards and inwards to the head of the femur. X-rays showed that neither wire was in a satisfactory position, one having slipped in front and the other behind the head of the bone. This procedure had to be twice repeated before the pin could be placed in position satisfactorily. The flanged Johansen pin was inserted through an incision about four inches long, the central hole in the pin being passed over the right wire guide and driven home. The last set of X-rays taken while the patient was on the table (actually the sixth

in point of number) showed the final position to be satisfactory. The spinal anæsthetic did not last long enough, and a little general anæsthetic by open ether was given from time to time at critical stages of the operation, the patient himself begging that he should not be rendered unconscious.

The circumstances of this operation were noteworthy. In spite of the utmost care in the matter of orientation the wires seemed obstinately to refuse to go where they were wanted, and yet everything was done with calm deliberation and utmost precision, without hurry or flurry. It was difficult and disappointing for an operator, who would naturally have liked everything to go quickly and smoothly. There was, however, no sign of irritation either by word or gesture, nor was there the slightest suggestion of abandoning the attempt until success had been achieved. We saw a number of other cases in which this operation had been done with complete success at varying periods after the accident. On return to his bed the patient's leg was elevated on a Braun's splint, with adhesive plaster traction and suspension of the toes. This was to be maintained for a week, when a plaster cast would be applied and fourteen days after the operation he would be allowed to walk.

THE TREATMENT OF CERTAIN TYPES OF CASE SEEN IN THE RECEIVING-ROOM AND ON THE WARD ROUND

The Setting and Fixing of Recent Closed Fractures—Each case is immediately X-rayed and anæsthetized by local infiltration, brachial plexus, or spinal anæsthesia. In fractures near the joints with no apparent displacement, an attempt



FIG. 224.—Fracture of femur treated by elevation and traction.

is made to produce displacement and X-ray examination is repeated. In this way a good idea is obtained of the liability to displacement. The fracture is then reduced, fixed by manipulation and skeletal traction, and again X-rayed. When satisfactory it is fixed in an unpadded plaster cast, the skeletal traction usually

remaining. The limb is always put up in an elevated position—the leg on a Braun's splint, the arm on an aeroplane splint (*Figs 224, 225*). The next day, and then daily afterwards, all joints of the limb not fixed by plaster are moved by active contraction of the muscles. In closed fractures of the leg below the knee a few days after the setting a walking-iron is incorporated in the cast, and the patient



FIG 225—Fracture of humerus Traction on an aeroplane splint

gets up daily. No crutches of the ordinary kind are allowed, but in the first days of walking four-legged hand crutches are used until the patient can support himself with sticks. Patients with fractured femurs begin to walk in casts with walking-irons from six to eight weeks after injury. In all cases of lower-limb fractures, when the plaster cast is removed an Unna's paste dressing is applied from the toes to the knee, and in this way œdema is prevented. We actually saw a great number (over 100) cases of closed fractures of the lower limb, without ever seeing

any swelling or œdema round the ankle. No case of fracture had any stiff joints apart from those where the joint itself had been involved.

The formula for the treatment of recent closed fracture is, then: (1) Accurate setting and X-ray control, (2) Uninterrupted fixation until bony union is firm, (3) Elevation of the injured limb, (4) Active exercise of the muscles and proximate joints, (5) Natural use of the limb as soon as union allows.

Wounds and Open Fractures (*Figs 226, 227*)—Wounds of the skin and soft parts receive the same treatment whether there is an associated fracture or not. There is no washing of the limb or even of the skin round the wound. Exuberant hair is cut away by scissors or dry shaving, and the parts are freely painted with iodine. Local anæsthesia is employed, either infiltration round the wound, or of the regional nerves. Brachial plexus or spinal anæsthesia is used for the more severe type of case. The edges of the skin and damaged surface of the soft parts are excised.

The cleansing is mechanical, not by chemicals or washing. This mechanical cleaning by the careful excision of damaged tissues requires great time and patience. The patient illustrated in *Fig 228* had a machinery accident in which his arm was caught in a revolving belt. The wound toilet in this case occupied close upon three hours, and a general anæsthetic was necessary before its completion. The patient who had had this injury was seen by us a year later, with a very useful arm.

If the bone is fractured, all fragments of any size are preserved and dirty surfaces are cleaned by nibbling forceps, not by mere scraping with a spoon. No fixation or suture of the bone is permitted, in fact no ligatures or sutures of any kind are buried in the wound unless this should be necessitated by the injury of

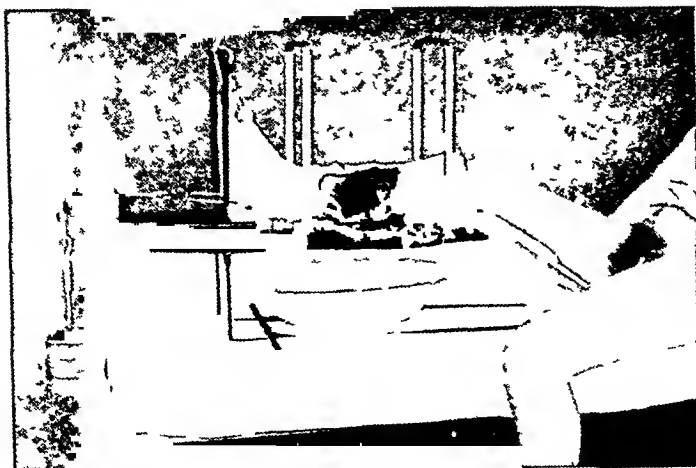


FIG 226 —Open fracture wound treated without any dressing



a



b



c



d

FIG 227 —Open fracture of tibia and fibula resulting from a run-over by a heavy lorry *a, b, c*, Condition on admission, *d*, X-ray taken four months later when the patient was able to return to work

a considerable blood-vessel or the division of a nerve-trunk. The skin is sutured if this can be done with not more than moderate tension. The fractured limb having been put up in the usual way (traction, plaster-of-Paris), the sutured wound is left quite uncovered and exposed to the air. Contamination by dust or flies is avoided by the use of a single layer of muslin arranged as a cage over, but not in contact with, the limb. If the wound heals by first intention, the silk stitches are left in place for one to six weeks. If the wound breaks down, or if there has been

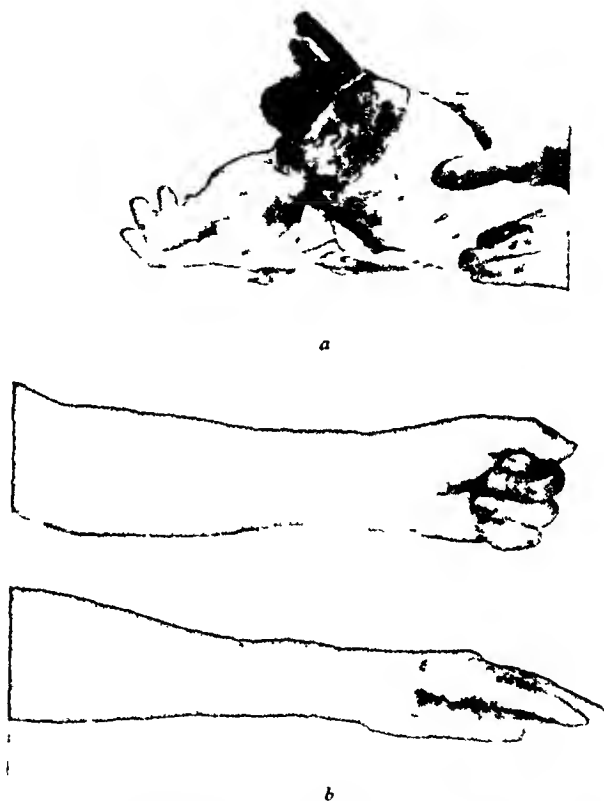


FIG. 228.—Open fracture of radius and ulna resulting from a machinery accident. *a*, Condition on admission, wound toilet took nearly three hours. *b*, A year later—condition when we saw the patient—almost perfect function.

a skin defect from the first, this is allowed to heal under a scab. We saw upwards of fifty cases of wounds, most of them associated with open fractures, and in not one was there any evidence of spreading infection, either local or constitutional.

Dr Bohler lays no claim to originality in the matter of not washing wounds, of using no dressings, or of applying plaster casts without padding. In regard to the last two points, at first he was forced by the necessities of the War to dispense with either dressings or padding, and having used these simple methods, he became convinced of their superiority.

Fractures of the Spine (Fig. 229).—These form a very important and interesting group of cases. After exact frontal, lateral, and sometimes oblique X-rays have been taken, the patient is kept at rest for a few days before reduction unless there is paralysis, in which case reduction is done at once. Local

(paravertebral) anæsthesia is given by injecting 5 per cent novocain by the side of the affected vertebral bodies. The reduction is made by hyperextension of the spine, the head and arms resting on a high table and the legs on one much lower. The rectification is checked by fresh X-rays, and if necessary the hyperextension is increased. The posterior prominence (gibbus) disappears as rectification is effected. The bony points of the pelvis are protected by wool stuck on by mastisol, the trunk is enveloped in a piece of stockingette, and the plaster case applied. Within three days to a week the patient is allowed up, and it is almost incredible how quickly he is able to carry out the exercises prescribed for him. At first these are only active movements of the arms and legs, but at a later period weights are carried on the head, beginning with 5 kilos and increasing to 40 kilos, whilst trapeze gymnastics are finally performed (Fig 230)

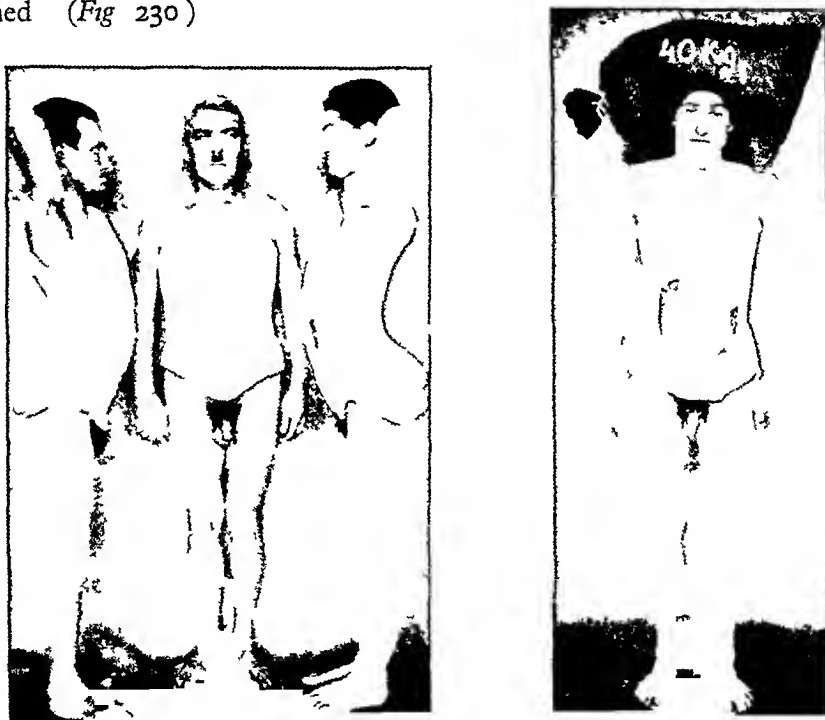


FIG 229 —Group of cases of compression fracture of spine in plaster. Note the hyperextension (lordosis).

There were actually five cases of compression fractures or fracture-dislocations of the spine as in-patients on the occasion of our visit. One, a case of fracture-dislocation of the cervical spine, was awaiting reduction, three cases were still in plaster, and one had had his plaster removed. All were men, and they occupied one ward. This principle of putting similar cases together was always carried out as far as possible. Each group seemed to form a happy family, or school class, the 'old boys' encouraging the 'freshmen'. All are desperately keen to do their lessons well, and the repetition of the exercises, individually and as a class, before the visiting surgeons, at least once a week, is an event which they evidently enjoy. Dr Bohler says that the treatment of injuries, and especially of fractures of the spine, is one part physical and three parts psychical.

The same spirit of happy enjoyment pervades the whole hospital, and among the 120 in-patients we saw only one or two depressed or unhappy, these being women recently admitted with head injuries. There were three cases of pulmonary infarct from embolism, all in stout middle-aged patients with fractures. This complication had occurred in all three patients within twenty-four hours, and we were told that this is their usual experience—namely, that a year or more will pass without a single case and then several cases will occur at the same time. It was suggested that meteorological conditions may account for this. Although accidents of all kinds are admitted, those involving thoracic and abdominal viscera constitute only about 2 per cent, and we saw only one case of appendicitis, one hernia, and one abdominal wound.

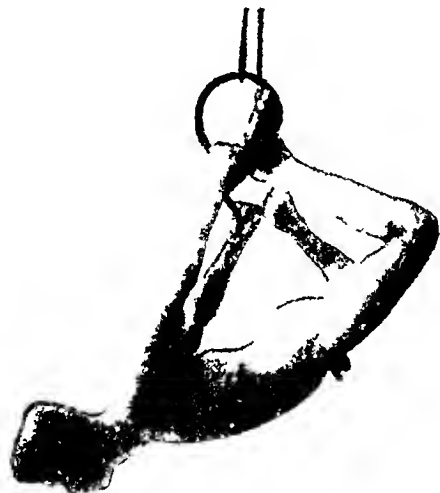


FIG 230—Exercises by man three months after compression fracture of spine

All patients who have been discharged from the hospital and have not yet returned to work are seen by Dr Bohler himself once a week, this inspection taking place on the ground floor of the large building. Every case is followed up until the final result is achieved.

The physiotherapeutical department is conspicuous by its absence. Except for some gymnastic appliances and those necessary for Swedish drill there is nothing of this sort. Massage and passive movement are forbidden in all recent injuries, and if proper treatment has been carried out, they are unnecessary at later stage.

The most prejudiced or sceptical observer cannot fail to be impressed by a visit to Dr Bohler. In matters of technical detail there must always be room for differences of opinion. Particularly in regard to the treatment of recent fracture cases by operative methods, it would seem to us that Bohler has an aversion that almost amounts to superstition. Also in cases of pseudarthrosis the modern bone-grafting operation might find a much larger place. But the broad principles of the work—meticulous care in observation, treatment, and records, unremitting hard work in carrying out the treatment, enthusiasm for ideals, originated by the Chief, loyally followed by the team, and happily accepted by the patients, and, most convincing of all, the acid test of results—these all defy criticism.

EXPERIMENTAL SURGERY

THE SECRETION OF MUCUS BY THE EPITHELIAL CELLS OF THE GALL-BLADDER, AND THE EXPERIMENTAL PRODUCTION OF MUCOCELE

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THE descriptions of the epithelium of the gall-bladder given in standard text-books of histology are woefully scanty, in nearly all instances slightly inaccurate, and sometimes notably untrue. For instance, Maximow (1933) states definitely that the gall-bladder does not secrete mucus, Mann (1928), Jordan (1927), and Cajal (1933) make no mention of mucus granules in the cells, the tenth edition of Schafer (1916) contains a drawing taken from Sommer (1909) showing the granules, but does not mention them in the text, a later (twelfth) edition leaves out even this picture. A very good description is given by Pfuhl (1932) in von Mollendorf's expensive text-book, but even this account contains several minor inaccuracies.

MATERIAL

I have examined the gall-bladders from a large number of cats, and from several rabbits, guinea-pigs, goats, sheep, pigs, and dogs. At the beginning a number of different fixatives was tried out, and as a result of these experiences Helly's fluid was used as a routine.

Normal human material is very difficult to obtain. I have examined gall-bladders removed at post-mortem as early as one hour after death, but in every case the mucosa was largely degenerated. This rapid degeneration after death is well recognized, Boyd (1922-3) gives some evidence that it is produced by the bile, since gall-bladders kept in water did not degenerate as rapidly as those kept in bile. By the kindness of Dr. Carleton, of the Histology Department at Oxford, I obtained a number of sections of a normal human gall-bladder removed at operation. Further, it seemed to me probable that most surgeons remove a normal gall-bladder occasionally—a few operators even admit this—and I therefore arranged to have a portion of every gall-bladder removed over a certain period in the Sheffield hospitals fixed in Helly's fluid immediately after removal from the patient and long before it would normally be placed in fixative. This has proved a useful method. Of fifty such gall-bladders two appear to be normal histologically as they did macroscopically, and the clinical history does not seem to demand a diagnosis of any lesion in the organ. I do not suggest that this is necessarily a true proportion for all operations, it is possible that the surgeons were influenced to some small extent by the knowledge that I desired to collect normal material.

The other cases in this series provide well-fixed examples of inflammatory changes of varying kinds and degrees. Besides these I have examined a large number of gall-bladders sent in for routine examination. These were usually fixed in 10 per cent formalin, a bad fixative since it causes swelling and bursting of mucin granules. Furthermore, the material is seldom put into the fixative until some time after removal, so that it is very unusual in such specimens to find an intact mucosa. From the point of view of the epithelium such material is useful only in studying the incidence of such structures as 'Luschka's ducts' and meta-plastic gland formation.

MUCUS SECRETION

The mucosa of the gall-bladder is covered by a single layer of tall cylindrical or prismatic cells, having a distinct resemblance to the intestinal epithelium from which they are derived during development. There is no essential difference between the mucosa of the gall-bladders of men, cats, dogs, goats, sheep, and pigs (*Fig 231*).

The epithelial cells of the rabbit's mucosa are, however, distinctive, in a manner similar to their intestinal epithelial cells (*Fig 232*). The cells are more darkly staining, much narrower, have their nuclei closer to the free border, and do not normally show mucin granules.

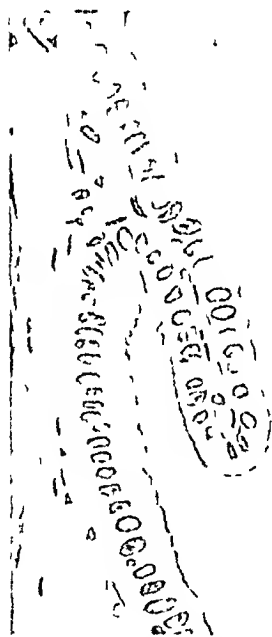


FIG 231—Normal cat mucosa. Fixed Helly's fluid. Stained haematoxylin and eosin. Drawn as seen under $\frac{1}{2}$ in objective.



FIG 232—Normal rabbit mucosa. Similar fixation, staining, and magnification as *Fig 231*.

In preparations fixed in Helly's fluid definite clearly outlined granules can be stained in the cells with mucicarmine. Many other fixatives, and in particular 10 per cent formol-saline, cause the granules to swell so that only a diffuse staining is to be obtained. Many writers—e.g., Pfuhl (1932) and Winkenwerder (1930)—state that a distinct red staining is not obtained with mucicarmine but only a pink coloration. I find that with good fixation and carefully prepared mucicarmine bright red staining of the granules is to be seen regularly in all my material except in the rabbit, for whose tissues Helly's fluid is possibly not a good fixative. More

consistent results are to be obtained with mucicarmine made according to Southgate's modification (1927), but the original Mayer's formula has worked well on occasions. In making the stain it is essential to follow the times given very closely, i.e., boiling must be for two minutes by the stop-watch. Metanil yellow used as a counterstain after hæmatoxylin and mucicarmine gives prettier pictures with the low powers of the microscope but tends to diminish the definition at high magnification.

The granules appear in that region immediately above the nucleus occupied by the Golgi body. From here they extend towards the free surface of the cell, sometimes in rows, and generally spreading out slightly fanwise so as to be closer to the lateral border at the summit of the cell. Granules are never seen below the level of the top of the nucleus in normal material, nor are they found reaching quite to the lateral borders of the cell. At the free border of the cell this mucus secretion can sometimes be stained passing through the pores or canals of the cuticle, more frequently tiny globules of secretion are visible sitting on the border after emerging from the cell.

In occasional cells containing only a relatively small amount of mucus, granules are found immediately above the nucleus only, or with also a few granules near the free border but at a distance from the supranuclear group (*Fig 233*). This appearance suggests that the granules may be formed in relation to the Golgi body as described by Florey (1932) in mammalian goblet cells.

It is interesting to note that Erdmann (1931) found granules staining with mucicarmine in her tissue-cultures of gall-bladder epithelium. The granules were not very numerous, and the cells contained about the same number whether they came from a ten days' or a fifty-six days' culture.

As is to be expected, not all the cells are to be found at the same functional phase in any given organ. Some are filled with secretion, others are practically emptied, and all stages in the formation and discharge of this secretion are to be found. Such variation is not so much in definite areas of the surface as diffusely scattered. Thus a cell so grossly swollen with mucus granules as to bear some resemblance to a goblet cell may be flanked on either side by thin cells almost devoid of granules. Only a small proportion of cells in a normal gall-bladder, however, vary greatly from the average appearance described above. Cells containing large quantities of mucus may be mistaken for goblet cells on superficial examination, and their nuclei tend to be pushed closer to the base of the cell and to be flattened like an orange. The occurrence of such cells in small groups in the gall-bladder of a dog led Cutore (1906) to describe them as a peculiar form of intra-epithelial gland formation. In a later vigorous article (1910) he accused Jurisch (1909) of publishing similar illustrations without acknowledgement and with a misinterpretation of their significance. Jurisch's reply (1910) is simple and effective: "each cell of the epithelium can secrete and does so to a variable extent

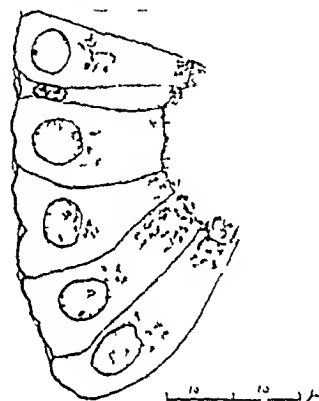


FIG 233—Normal human epithelial cells. Fixed Helly's fluid. Stained hæmatoxylin, mucicarmine, and metanil yellow. Drawn as seen through oil-immersion lens. Shows mucin granules in region of Golgi bodies and near free surface of cells, also striated border appearing as row of short hairs. The second cell from the top is a Stützcell.

according to its position—it does not advance our understanding of the secretory process to give special names to those cells which at the moment of fixation are secreting strongly”

Rather more common than the very swollen cells are others of a thin rod-shaped or whip-like form described at length as “Stiftzellen” by many writers

They are mentioned by Sudler (1901) and Aschoff (1905), whilst Shikunami (1908) found them in his material in varying numbers. Jurisch (1909) described them as very long thin cells having a darkly staining cytoplasm and with streak-like nuclei that might be indented or cornered. Both of the latter writers take the view that the secretion is formed in the ordinary epithelial cells, which with increasing fullness change into true goblet cells, the secretion then bursts through the cuticle and the emptied cell becomes a “Stiftzell”. While Shikunami believed that the

“Stiftzellen” were then freed and thrown off, Jurisch thought they could recover and that the functional cycle could start again. Winkenwerder (1930) described these cells and their staining characteristics, and traced the stages of their formation by the extrusion of mucus granules from the more usual epithelial cells. He found also that these cells exhibited a greatly augmented permeability for potassium ferrocyanide and ferric ammonium citrate. Although it is quite possible that these cells at the end of the secretory phase have a greater capacity for absorption than those actively forming secretion, his experiments are by no means conclusive since the chemicals used are definitely toxic. These “Stiftzellen” are to be found in varying numbers in all my material, occurring usually singly but occasionally in groups of three or four. Only a few of them are, however, completely devoid of granules, most of them show a few granules towards the free surface (see Fig 233). I believe that they do not represent a different type of epithelial cell, but only a particular functional phase.

I have performed some experiments to demonstrate the actual secretion of mucus by the epithelial cells of cats (Figs 234, 235). Under ether anaesthesia a small portion of the gall-bladder wall was removed and fixed immediately in Helly's fluid to serve as a control since the amount of mucus present varies from animal to animal. The opening in the bladder was sewn up and a solution of mustard-oil in olive-oil

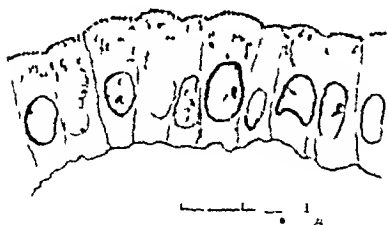


FIG 234—Normal cat epithelium. Similar fixation, staining, and magnification to Fig 233. Shows mucin granules and striated border.

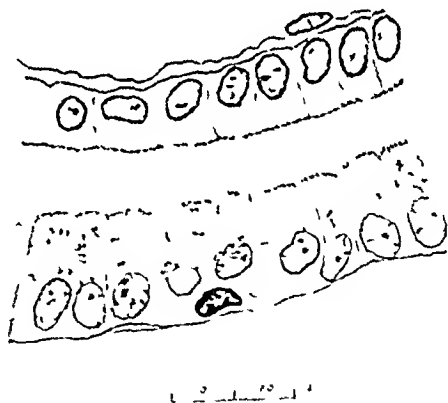


FIG 235—Normal cat epithelium. Similar fixation, staining and magnification to Fig 233. Variation in mucin content in different areas. The two portions of epithelium illustrated become continuous further to the right in the actual specimen. (Note.—I have been unable to draw cells containing more than a moderate number of granules. Many cells contain a far greater number than those figured here.)

introduced into the organ, after this the abdomen was closed and intravenous chloralose given. At the end of two hours the cat was killed, the gall-bladder removed immediately and portions of it fixed. It was found, as might be expected, that the epithelium was a very fragile structure and that mustard-oil solutions of a strength innocuous to the stomach or intestine produced marked damage. Weaker solutions (2 drops in 25 c c) produced no evident naked-eye or microscopical damage, but a very definite evacuation of the mucus granules was found in sections. It would appear, then, that the epithelial cells can be provoked to secrete by a mild experimental irritation. It is improbable that mustard-oil has any specific action beyond its irritative properties, and it seems likely that the operative procedures played some part in the production of the phenomena. Nevertheless, similar experiments using plain olive-oil did not give any marked evacuation of mucus. Evacuation of mucus has also been produced in cats by intravenous injections of pilocarpine in large quantities ($\frac{1}{2}$ c c of 1-1000 solution every fifteen minutes for two to three hours).

In naturally occurring inflammations in the human gall-bladder the mucus content of the epithelial cells varies considerably. Occasionally a mild acute inflammation results in the cells containing a greater amount of secretion than normal, and more mucus is found lying on the free surface. This is a condition of "catarrhal cholecystitis", and is only rarely seen in histological preparations. More frequently acute inflammation produces a destruction of a large number of the epithelial cells, especially the more superficial ones, ulcers are thus produced, and there is an accompanying acute inflammatory exudate of sero-fibrinous fluid and polymorphonuclear leucocytes. The remaining epithelial cells in these cases of "ulcerative or purulent cholecystitis" usually contain only a few granules of mucin. In chronic or recurrent inflammations of the gall-bladder the main features of the epithelial layers are the occurrence of hyperplasia with prolongations into the wall of the organ, and of metaplasia. Generally the amount of mucus secretion is increased, but there is considerable variation from case to case, and frequently in different areas in the same case. In the condition described as 'mucocoele' or 'hydrops' of the gall-bladder, I have usually found marked flattening of the epithelial cells which contain only traces of secretion. I have not, however, any really well fixed material—most of my specimens have been obtained post-mortem, and the others are rather inadequately fixed in formalin. Bauer (1931) and Bauer and Hakkı (1932) report cells filled with mucin granules—a "mucoid transformation"—in their specimens.

EXPERIMENTAL PRODUCTION OF MUCOCELE OF THE GALL-BLADDER

Numerous experimenters have endeavoured to produce in animals a condition similar to the mucocoele or hydrops found occasionally in man as a result of obstruction at the neck of the gall-bladder. Such an obstruction is commonly due to the impaction of a solitary stone in the neck of the gall-bladder, but may be produced in other ways. Riedel (1903) considered that the condition had a purely mechanical origin, but Aschoff (1905) thought that simple obstruction was insufficient and that an added infection was needed. He found that in dogs an aseptic tying or resection of the cystic duct did not produce such a condition, but resulted in an

atrophy of the gall-bladder Mocquot (1909) saw a temporary mucous distension as a result of such experimental interference, but this was followed by definite contraction of the organ Mignot, Businco (1917), Borgi (1921), Agrifoglio (1923), and Petrescu (1924) failed to produce anything more than a temporary distension, followed invariably by a contraction of the organ with a progressive atrophy of the mucosa and concentration of the contained bile Galli and Vecchi (1928) tried tying the cystic duct, introducing a sterile foreign body (human gall-stone) into the gall-bladder, infecting the gall-bladder with typhoid bacilli, and combinations of these procedures in none of their thirty-one dogs did they produce a condition comparable with hydrops in the human

Toida (1920), experimenting on dogs, cats, and rabbits, found that simple aseptic tying of the cystic duct, with or without a tying of the cystic artery or the introduction of a foreign body, did not produce a condition of hydrops but only a sclerosis or contraction of the bladder and a thickening of the contained bile However, the introduction of bacteria of feeble virulence into such an obstructed gall-bladder provoked a catarrhal type of inflammation and he was thus able to produce what he described as a typical hydrops Bacteria of a higher virulence produced a purulent inflammation

Hunt, Davis, and Boyden (1931) tied the cystic duct in cats, and examined the results at intervals up to a fortnight They found that only 50 per cent of the gall-bladders showed a normal mucosa, in 20 per cent it was oedematous and in 30 per cent definite inflammatory changes were present They state that such operations in cats are always followed by some degree of cholecystitis, which ranges from the mildest inflammation to a hydropic distension of the organ, although there is no evidence of infection of the cystic bile They conclude that hydrops of the gall-bladder may occur in the absence of infection, and believe that it is due to moderate interference with the blood-supply, to which interference the irritating action of stagnant bile may be a contributory but not essential factor A fair criticism of their conclusions is that they observed only the immediate and early results of occlusion Earlier writers have noted that there is sometimes a temporary distension but that a true mucocoele does not eventuate under these conditions The high proportion in which inflammatory changes were demonstrated histologically is noteworthy in view of the findings of Andrews and Hrdina (1930) and Andrews, Schoenheimer, and Hrdina (1932), who state that it is very difficult to produce stasis of any sort in the gall-bladder of the dog without the prompt onset of sepsis When the cystic duct is tied and the gall-bladder needled, severe degenerative changes promptly appear owing to infection arising in the occluded, traumatized organ The bile, which was sterile, promptly becomes infected by great numbers of organisms, and microscopical examination of the wall shows a marked onset of a moderately severe inflammatory process

Bauer (1931) confirmed the findings of Toida (1920) that it is impossible to shut off the gall-bladder for any length of time by introducing a foreign body into the lumen such bodies wander readily either into the fundus of the bladder or down the bile-ducts He therefore tied the cystic duct in dogs, generally including the cystic artery in the ligature Provided the vesicle was not detached from the liver he found no alteration directly attributed to the tying of the cystic artery, whose functions were taken over by anastomosing vessels When the gall-bladder was emptied of bile and washed out with saline an immediate secretion of mucus

occurred, at first small in amount, clear and fluid, then increasing in quantity, becoming more and more dense and containing greyish-white floccules. No distension of the bladder occurred, however, it filled up slowly to a normal size. At the end of three months the condition was well marked. The mucosal cells, particularly those at the fundus, showed an increased content of mucus granules staining deeply with mucicarmine. After this period there occurred a regression, the epithelium ceased to secrete mucus, the cells tended to become flattened and to contain few granules staining with mucicarmine. At a still later period (eight months) the contents were found to be again fluid, suggesting a re-absorption or possibly a destruction of the mucus. When the gall-bladder was not emptied of its bile such a sequence could not be produced. Instead there was a degeneration of the epithelium, attributed to the toxic action of the retained bile, and mucus-producing cells were rare. The bile became greatly concentrated, but there was very little evidence that anything but water was absorbed. The introduction of a sterile gall-stone into the occluded, washed-out gall-bladder appeared to augment the intensity of the production of mucus, but again there was no distension. Inoculation of a suspension of *B. coli* of attenuated virulence into such a bladder gave a rapid distension of the organ with a clear mucoid fluid comparable with that found in a human mucocele. This result could only be obtained provided the cystic artery was intact, a fact noted previously by Toida (1920). Bauer did not follow any of these cases for more than a fortnight, at the end of which time *B. coli* could still be cultivated from the distended organ. These experiments are also recorded in a paper by Bauer and Hakkı (1932), but with fewer details. They insist that a true mucin is produced by the epithelial cells of the gall-bladder in dogs and man.

AUTHOR'S EXPERIMENTS

In a number of experiments on the gall-bladder of the cat, a few have some bearing on this problem. All the experiments were performed under ether, and aseptic precautions were taken throughout. In every case the cystic duct was occluded by a ligature of silk, the cystic artery being sometimes included in the ligature but left free whenever possible. No evident difference was found at any time as a result of inclusion of the artery, there is apparently a sufficient collateral circulation from the surface of the liver to which the gall-bladder is usually closely applied in the cat. The occlusion of the cystic duct was tested when the animals were eventually killed, and in all cases the duct appeared to be completely shut by the ligature. In one case, however, after 53 days the contents of the bladder appeared to be normal bile. Although it could not be demonstrated, it appeared likely that there was an accessory duct or an anastomosis via the small tributary described by Rous and McMaster (1921). This experiment was disregarded. In two cases in which the cystic duct was occluded for six months, the common bile-duct was distinctly dilated.

In experiments where the only interference consisted of tying the cystic duct, a condition that could be described as a mucocele did not develop. Such gall-bladders were examined 4, 48, 90, and 181 days after the operation. The first two were of normal size, the third about half normal, and the last quite small. The contents were in all cases thick dark bile, which in only one case (48 days) appeared to the naked eye to be mixed with mucus. Definite histological evidence

of inflammation was to be found in all except the last. In short, these findings agree with all the previous published results—simple obstruction of the cystic duct does not lead to the formation of a mucocele. In another case an anomalous result was obtained after simple tying of the duct. This animal was killed 28 days after the operation, the vesicle was definitely but not greatly distended and appeared normal on its external surface. The contents were found to consist almost entirely of material resembling white of egg, whilst only at the fundus was there some pale yellow opaque material applied to the mucous membrane. There was histological evidence of a subsiding inflammation, but no organisms could be found in smears or sections. This experiment does not fit in with those recorded above or with previously published ones. Judging from the small amount of bile pigment present in the bladder after four weeks, it seems probable that the organ contained only a very small amount of bile or only very dilute bile at the time of operation. No other experiments show absorption of large amounts of bile in so short a time, and there was no microscopical evidence of such an absorption in this case. If this experiment is to be included in this series it should be placed rather amongst those in which the gall-bladder was emptied of its bile. I prefer to record it as an anomalous result and not to include it with either group.

In other cats, after ligating the cystic duct, the gall-bladder was emptied through a fine needle, washed with sterile saline, and left partially filled with saline. One such after 29 days was small, contained only a little mucus, and showed no histological evidence of inflammation. One left for 88 days was found to be enlarged and tense, it was distended with very thick sticky material coloured a golden brown by altered blood pigment (the wall had been deliberately damaged with forceps at the operation) no organisms could be detected, but there was definite histological evidence of a mild inflammatory reaction. A third gall-bladder after 187 days was enlarged to about twice the normal size, it contained a clear, viscid fluid like egg-white, which at the fundus was rendered opaque by numerous glistening particles of cholesterol. No histological evidence of inflammation could be found in the body of the bladder but the appearances at the fundus suggested a continued mild inflammatory lesion, no organisms could be found in smears or sections.

Attempts to produce a mucoid dilatation by injecting *B. coli* obtained from cat faeces were uniformly unsuccessful. A purulent cholecystitis resulted in every case.

DISCUSSION

It is possible, though difficult, to produce in the gall-bladder of the experimental animal a condition comparable with human mucocele. The presence of bile in the occluded bladder prevents this happening, presumably because the inspissated bile tends to produce a destruction of the epithelial lining. In a similar manner infection by organisms having an appreciable virulence does not produce such a condition, there is a purulent inflammation with destruction of mucosal cells. For the development of an experimental mucocele the gall-bladder must be emptied of all but traces of bile and a very mild inflammation be initiated. That emptying of the bile after occlusion is not of itself sufficient is shown in an experiment recorded above where after 29 days the bladder was small and shrunken, and there was no evidence of inflammation.

Toida (1920) produced the condition by injection of organisms of feeble virulence, and presumably evacuated the bile at the time of injection. Bauer (1931) produced a mucoid distension by washing out the occluded gall-bladder, but found that the condition tended to regress. He stated that there was no inflammatory reaction as a result of this procedure, but in view of the findings of Andrews and Hrdina (1930) it appears probable that a mild inflammation was set up as a result of infection at the time of the operation.

In the cases of mucocoele reported here there was in every case histological evidence of a mild regressing inflammation affecting part of the mucosa. It appears possible that in these cases a mild infection resulted from the operative interference, and that the consequent mild inflammation stimulated the epithelial cells to secrete large quantities of mucus and thus produce a mucocoele. The mucoid material distending the bladder is not purely mucus but a mixture of mucus and an albuminous material, as is readily seen in smears or sections. This is true of the contents of human mucocoeles, but commonly the proportion of mucus is less—an albuminous fluid containing mucus. Crystals of cholesterol may be found in the human fluid as in one of the cases recorded above. It appears probable that in the production of a mucocoele of the gall-bladder in man several factors must play a part: (1) Obstruction, commonly by a stone, (2) Absorption of the bile contained in the vesicle,* or its expulsion by a mucous secretion before obstruction is complete, (3) A mild continued inflammation, probably bacterial but with organisms of a virulence too low to produce a purulent reaction.

These conclusions agree with those put forward by Illingworth and Dick (1932): "the effects of obstruction of the cystic duct, however it is caused, depend on the degree and virulence of any present or potential infection. When the process is entirely non-inflammatory the gall-bladder gradually contracts and its contents become inspissated. When a very mild degree of inflammatory change is present, the first effect of obstruction is to cause a secretion of mucus, and the gall-bladder then becomes distended as a mucocoele, which may remain completely aseptic. When bacterial infection is present, the condition is that known as acute obstructive cholecystitis."

SUMMARY

The mucosa of the gall-bladder has a single layer of large columnar or prismatic cells thrown into folds of varying height according to the degree of distension of the organ. There are no essential differences in the appearances of the cells in men, cats, dogs, goats, sheep, pigs, or guinea-pigs. The individual cells vary considerably in shape, size, and intensity of staining according to their functional phase, but there is only one type of cell present in the normal epithelium. Within the cytoplasm are found granules of mucus staining brightly with mucicarmine. Their number varies greatly in different gall-bladders and in different parts of the same organ, but they are always present in normal tissue. It is suggested that the granules are formed in the region of the Golgi body. Experimental evidence is given that the cells can be emptied of mucus by a mild irritation.

* That bile pigment can under appropriate conditions be absorbed from the occluded gall-bladder has been shown on several occasions, and examples were found in the above experiments.

The epithelium of the rabbit's gall-bladder is readily distinguished from that of the other animals examined. The cells are more darkly staining, much narrower, have their nuclei closer to the free border, and do not show mucus granules.

Experiments are described to show that it is possible to produce a condition of mucocoele in the gall-bladder of the cat by tying the cystic duct and emptying the vesicle of its contained bile. A factor in the production of this experimental mucocoele is possibly a mild inflammation incidental to the operative procedures.

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BIBLIOGRAPHY

- AGRIFOLIO, M, *Chin med ital*, 1923, *lv*, 1.
 ANDREWS, E, and HIRDINA, L, *Proc Soc Exper Biol*, 1930, *xxviii*, 116.
 ANDREWS, E, SCHOENHOFMER, R, and HIRDINA, L, *Arch of Surg*, 1932, *xxv*, 796.
 ASCHOFF, L, *Verhandl deut pathol Ges*, 1905, *ix*, 41.
 BAUER, R, "Récherches expérimentales sur les mucocèles vésiculaires", *Thèse de Strasbourg*, 1931.
 BAUER, R, and HAKKI, A. C., *Presse med*, 1932, *i*, 650.
 BORG, L. *La litasi biliaire*, 1921. Milan (quoted from Galli and Vecchi, 1928).
 BUSINCO, *Pathologica*, 1917 (quoted from Galli and Vecchi).
 CAJAL, S. RAMON Y, *Histology*, 1933. London.
 CUTORE, G, *Arch di anat e di embriol*, 1905, *v* (quoted from Cutore, 1910).
 CUTORE, G, *Anat Anzeiger*, 1910, *xxxvi*, 100.
 ERDMANN, R, *Arch f exper Zellforsch*, 1931, *vi*, 530.
 FLOREY, H. W., *Brit Jour Exper Pathol*, 1932, *iii*, 349.
 GALLI, G, and VECCHI, A, *Arch ital di Chir*, 1928, *xxi*, 298.
 HUNT, E. A., DAVIS, A. H., and BOYDEN, E. A., *Anat Record*, 1931, *lxix*, 295.
 ILLINGWORTH, C. F. W., and DICK, B. M., *Text-book of Surgical Pathology*, 1932. London.
 JORDAN, H. E., *Text-book of Histology*, 1927. New York.
 JURISCH, A., *Anat Heft*, 1909, *xxxix*, 393.
 JURISCH, A., *Anat Anzeiger*, 1910, *xxxvi*, 526.
 MANN, F. C., "The cytology of the biliary channels", in *Special Cytology*, 1928, 1. New York.
 MAXIMOW, A. A., *Text-book of Histology*, 1931. Philadelphia.
 MIGNOT, Quoted by Galli and Vecchi, 1928.
 MOCQUOT, "L'Etat de la vesicule biliaire dans les obstructions des voies biliaires", *Thèse de Paris*, 1909 (quoted from Bauer).
 PETRESCU, G. J., *Presse med*, 1924, *i*, 539.
 FRUHL, W., "Die Gallenblase und die extrahepatischen Gallengänge" in von Mollendorf's *Handbuch der mikroskopischen Anatomie des Menschen*, 1932, *v*. Berlin.
 RIEDEL, *Pathogenese der Gallensteinleiden*, 1903. Jena.
 ROUS, P., and McMASTER, P. D., *Jour of Exper Med*, 1921, *xxxiv*, 75.
 SCHAFER, E. A., *The Essentials of Histology*, 1916, 10th ed., 363. London (12th ed., 1929).
 SHIKINAMI, J., *Anat Heft*, 1908, *xxvi*, 555.
 SOMMER, A., *Verhandl der anat Ges*, *Anat Anz*, 1909, *xxxiv*, 148.
 SOUTHGATE, H. W., *Jour Pathol and Bacteriol*, 1927, *xxx*, 729.
 SUDLER, M. T., *Bull Johns Hopkins Hosp*, 1901, *xii*, 126.
 TODA, R., *Zentralb f Chir*, 1923, *xv*, 627. (Abstract from *Mitt a d med Fak d Kais Univ Kyushu*, Fukuoka, Japan, 1920, *v*, 131).
 WINKENWERDER, W. L., *Bull Johns Hopkins Hosp*, 1930, *lxvi*, 272.

SURGICAL EXPOSURE OF THE ŒSOPHAGUS

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RECENT advances in physical therapy and the continued mortality of surgical intervention have led many to suppose that the treatment of disease in the œsophagus should become the domain of the physical therapist and the endoscopist. The recent demonstration of a successful intervention for carcinoma of the œsophagus by Grey Turner has done much to stimulate interest in the surgical treatment of such disease. It is to be remembered that in many parts of the world treatment must remain surgical, and, even if it should prove that in certain cases physical

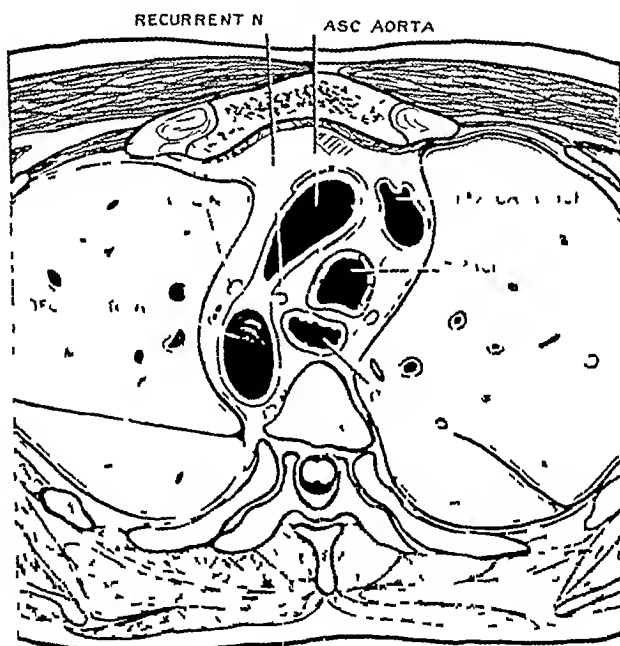


FIG 236 —A transverse section of the thorax at the level of the 4th thoracic vertebra, showing the relations of the œsophagus above the bifurcation of the trachea (After Eycleshymer and Schoemaker)

therapy is to be preferred, it is essential that surgery should be in a position to offer a less fatal alternative than is at present the case. Innocent tumours of the œsophagus are rarely diagnosed, and in most of the recorded cases diagnosis has only followed exploratory operation, it may be assumed that more frequent exploration might lead to the more frequent discovery of conditions for which surgery can provide a dramatic cure. Some cases of this kind operated on by Sauerbruch have recently been recorded by Krauss, and Ohsawa has also extirpated a myoma of the œsophagus with successful results.

The initial difficulty in œsophageal surgery is adequate exposure of the organ,

and this alone will be considered here. A series of transverse sections (Figs 236-238) give an impression of the apparently complex relations of the œsophagus,

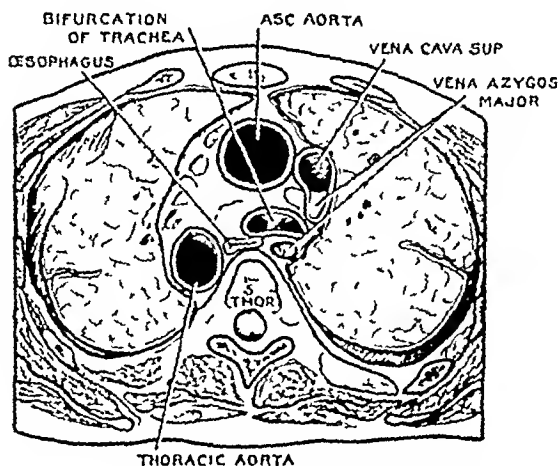


FIG. 237.—A transverse section of the thorax at the level of the 5th thoracic vertebra showing the relations of the œsophagus at the bifurcation of the trachea. (After Professor R. J. A. Berry.)

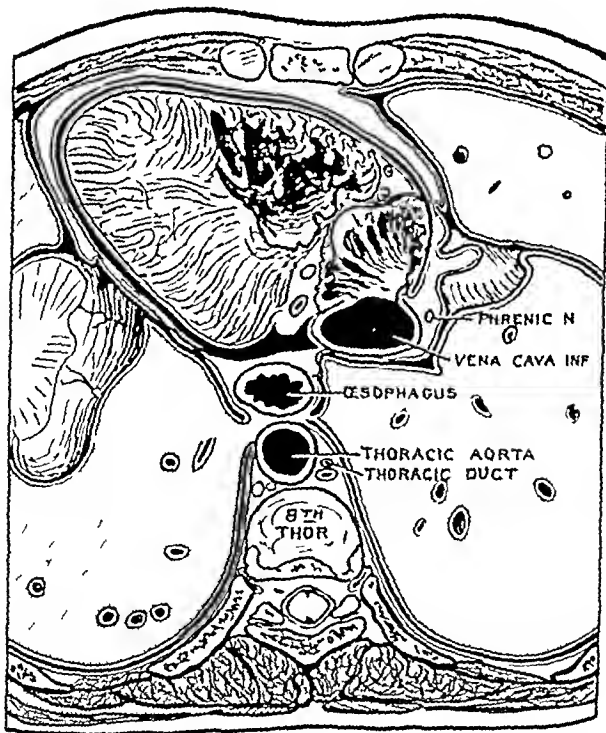


FIG. 238.—A transverse section of the thorax at the level of the 8th thoracic vertebra, showing the relations of the œsophagus below the bifurcation of the trachea. (After Eycleshymer and Schoemaker.)

but the main structures are easily identified if an exposure based on the lines laid down in this paper is employed. A series of dissections has been carried out, and

careful measurements have been made of the length of the œsophagus which may be displayed at various levels and the depth from the surface at which it lies. At the same time the copious literature of the œsophagus has been studied, and reference will be made to some papers which are of special assistance to those who contemplate a surgical attack on the organ.

EXPOSURE OF THE CERVICAL ŒSOPHAGUS

Exposure of the cervical œsophagus has been carried out in cases of carcinoma, pharyngeal diverticula, and for the extraction of foreign bodies.

Some early cases of excision of a carcinoma of the cervical œsophagus by Sauerbruch are reported in detail by Schelbert. One of these is of special interest as the growth extended into the upper part of the thoracic inlet. A somewhat similar case has been reported recently by Evans, and Kuttner has also recorded a successful case in some detail. In these cases the tumour had invaded the larynx, and this organ was extirpated as well as a segment of the œsophagus. Despite

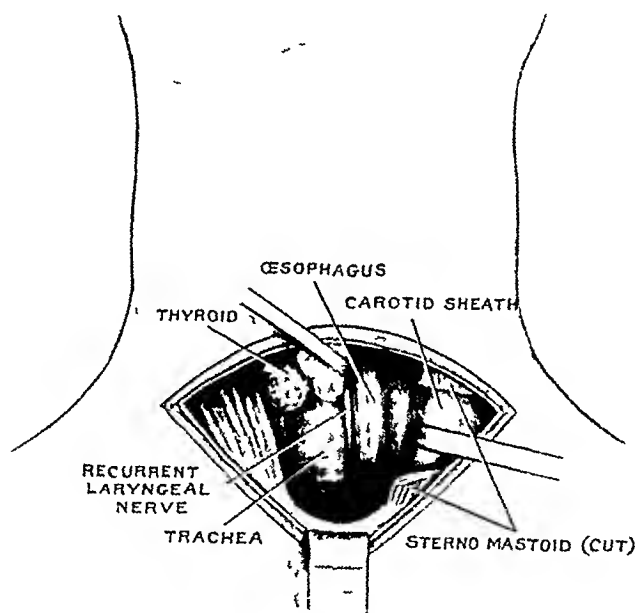


FIG. 239.—Exposure of the cervical œsophagus at the thoracic inlet through a curved incision and division of the left sternomastoid muscle. The left carotid sheath is retracted to the left, and the left lobe of the thyroid gland is retracted to the right.

this mutilation, the patients were able to talk, although in a low tone, and it is recorded by Evans that his patient was able to carry out her own shopping without difficulty.

There are very many accounts of operation for pharyngeal diverticula in the literature. One of the most comprehensive is by Wilkie and Hartley. Approach is by a longitudinal incision along the anterior border of the sternomastoid, and after division of the middle thyroid vein a lateral retraction of the carotid sheath affords exposure of the sac.

Modern endoscopy has rendered the classical intervention of cervical œsophagotomy for the extraction of a foreign body almost obsolete. A very complete account of the indications, the method, and the literature are given by von Hacker and Lotheissen.

Technique (*Fig. 239*)—The patient lies on his back, with the head fully extended and rotated to the right. An incision is made 11 cm. long, commencing at the left sternoclavicular joint and passing upward along the anterior border of the sternomastoid muscle. The anterior border of the sternomastoid is defined and the muscle is retracted outwards, in this way the intermediate tendon of the omohyoid muscle is exposed and retracted upwards. Ligation and division of the middle thyroid vein constitutes the next step of the operation. The carotid sheath is then exposed and retracted outwards, and the left lateral lobe of the thyroid gland, the sternohyoid, and sternothyroid muscles are identified and retracted inwards. The inferior thyroid artery is exposed in the lower part of the wound. The left recurrent laryngeal nerve is seen lying posterior to the lateral border of the trachea and in front of the œsophagus. The œsophagus, as it lies between the trachea and the vertebral bodies, is exposed in a length of 9 cm., at the level of the upper border of the manubrium sterni its depth from the surface is 6 cm.

In his operation for the collo-abdominal extirpation of the œsophagus Grey Turner makes a transverse incision in the neck and divides the sternomastoid muscle. In our dissections we found that this method enabled 11 cm. of the œsophagus to be exposed.

EXPOSURE OF THE THORACIC ŒSOPHAGUS

The thoracic œsophagus has been exposed in cases of carcinoma, benign tumour, diverticulum, and for the extraction of foreign bodies. Two routes are available for exposure—the mediastinal and the transpleural. Mikulicz was perhaps the first surgeon to make a determined attempt to exploit the surgery of the thoracic œsophagus, and, after repeated trials of the mediastinal route, he came to the conclusion that only the transpleural route could give adequate exposure. The chief modern advocate of the transpleural route is Sauerbruch. Enderlen, on the other hand, preferred the mediastinal route, and his account of the surgery of the posterior mediastinum is of the utmost value. Lilienthal still employs this approach, and it may be noted that one of the three cases in the world literature up to 1933 in which natural swallowing was restored after excision of a carcinoma was carried out by him. All authorities are agreed that the mediastinal approach is to be preferred in such cases as impaction of a foreign body necessitating œsophagotomy, in intervention for perforation or rupture of the organ, or in extirpation of small diverticula which have perforated into a bronchus. In this type of case it is a considerable advantage to be in a position to leave a large open wound for drainage.

The merits and disadvantages of the mediastinal approach are illustrated by a recent operation which one of us (L. O'S.) performed. The œsophagus was approached by a posterior mediastinotomy in the region of the lung root. A tumour was exposed which was adherent to the left bronchus, and separation could not be effected. The intervention lasted for one hour, and the wound healed by first intention. Only at autopsy some four months later was it disclosed that the tumour was actually a carcinoma of the left bronchus which had invaded the

œsophagus With a better exposure this diagnosis should have been established at the operation, but on the other hand the immediate post-operative recovery might have been more delayed

In exposing the upper thoracic œsophagus by the transpleural route Sauerbruch resects the second and third ribs from the sternal margin for 6 cm of their extent, incises the sternoclavicular joint and dislocates the sternal end of the clavicle, and finally resects a portion of the first rib The pleura is opened by a vertical incision and the œsophagus identified as it lies behind the trachea Barrett has recorded an operation by Romanis in which a diverticulum was removed which opened into the œsophagus just below the level of the azygos vein Approach in this case was made through an incision in the sixth intercostal space

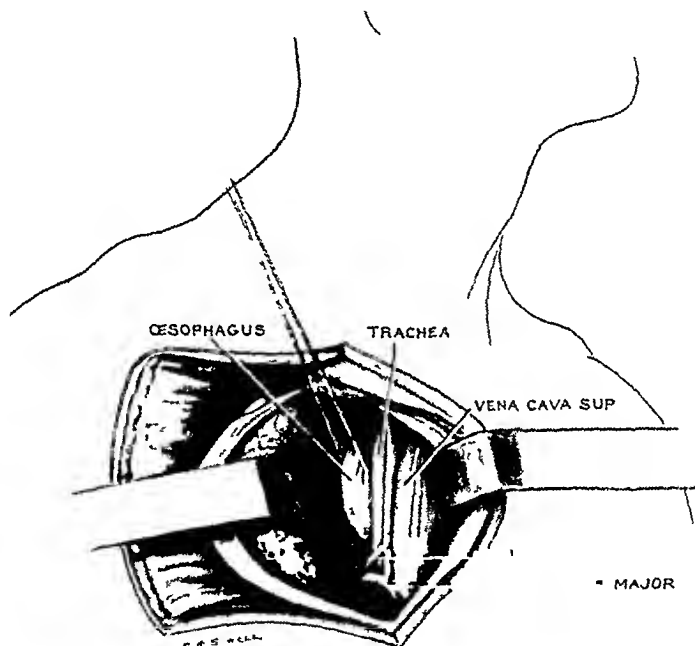


FIG 240—Exposure of the thoracic œsophagus above the bifurcation of the trachea by the right transpleural route The right lung is retracted outwards and the œsophagus is seen behind the trachea and crossed by the vena azygos major and the right vagus nerve

For his successful case of radical operation for carcinoma Torek made an incision along the whole length of the seventh left intercostal space The fourth, fifth, sixth, and seventh ribs were divided in the region of the tubercle and the pleura incised With this exposure an excision of the entire œsophagus from the diaphragm to the thoracic inlet was carried out under vision Kirschner has used the same interspace in displaying the lower œsophagus and the region of the cardia Division of the seventh costal cartilage and the left rectus permits of a wide exposure of the diaphragm, and if necessary this muscle is divided in a line stretching from the œsophageal hiatus to the periphery Hedblom and Zaaiger have employed a very similar approach, but these authorities consider that a preliminary resection of the lower ribs carried out some ten days before the final operation facilitates the subsequent exposure A left transpleural approach to the

lower œsophagus is also used by Sauerbruch, and this route was adopted by Eggers in his successful case

Gregoire has described in minute detail an extrapleural approach to the lower end of the thoracic œsophagus. He resects the twelfth rib on the left side, and after simple division of the tenth and eleventh ribs the pleura is separated from the costovertebral sulcus. The œsophagus is exposed between the aorta on the medial side and the lung, covered by the separated costal pleura, on the lateral side. By incising the diaphragm and so exposing the cardiac end of the stomach Gregoire has carried out an œsophagogastrostomy by this route.

Lilienthal enters the mediastinum after resection of the ninth rib on the left side. According to the exposure required, further ribs are excised after separation of the parietal pleura has been begun. Lilienthal mentions the descending thoracic aorta and the great splanchnic nerve as important landmarks in the initial stages of the operation.

Technique —

1 *Right Transpleural Exposure of the Upper Œsophagus (Fig 240)* —The patient lies on his back with the upper extremities extended by the side. An incision 10 cm long is made over the second rib from without inwards to the sternum. It is then carried vertically downwards over the sternum to the fourth costochondral junction and from thence outwards in the line of the fourth rib for 10 cm. This flap, consisting of the skin, superficial and deep fasciæ, and costosternal portion of the pectoralis major muscle and the pectoralis minor muscle, is dissected from the deeper structures and reflected outwards. The periosteum is reflected from the second and third ribs over 12 and 14 cm of their length respectively, and similar lengths of these ribs, with their costal cartilages, are removed. The internal mammary artery and veins are exposed in the inner part of the wound. An incision is made in the second intercostal space to open the pleural cavity. The right lung is retracted outwards and downwards and the following structures are identified. The superior vena cava with the right phrenic nerve and accompanying blood-vessels is seen in the medial part of the wound. The vena azygos major is seen arching over the root of the lung to join the superior vena cava. The trachea is easily identifiable, and the right vagus may be found in a triangle bounded by the trachea, the vena azygos major, and the superior vena cava. The vena azygos major is divided and the mediastinal pleura incised to expose the œsophagus as it lies behind the trachea and above the right bronchus. In this way 8.5 cm of the œsophagus may be exposed—the upper limit being the thoracic inlet and the lower limit the bifurcation of the trachea. The exposed œsophagus lies at a depth of 7.5 cm.

2 *Right Transpleural Exposure of the Lower Œsophagus (Fig 241)* —The patient lies on the left side, with a pillow under the ribs and the right arm held by an assistant as in the operation of paravertebral thoracoplasty. An incision is made over the sixth rib from the costal cartilage to the inferior angle of the right scapula. A subperiosteal resection of some 26 cm of the sixth rib is then carried out. An incision is made in the sixth intercostal space and the pleural cavity opened. After lateral retraction of the right lung the following structures are seen anterior to the œsophagus—the bifurcation of the trachea, the left bronchus, the pericardium, and the diaphragm. Posteriorly are found the vena azygos major and the thoracic duct. Near the diaphragm the œsophagus becomes anterior to the descending thoracic

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aorta, and above it is crossed by the vena azygos major. In this way the œsophagus is exposed between the lung root and the diaphragm in a length of 14 cm. Division and ligation of the azygos vein enable the organ to be followed up to a rather higher level, so that in all 16 cm may be exposed. The œsophagus lies at a depth of 12 cm.

3 *Left Transpleural Exposure of the Lower Œsophagus* (Fig 242)—The patient lies on the right side with a cushion beneath the ribs and the arm held by an assistant as for a paravertebral thoracoplasty. An incision is made over the sixth rib,

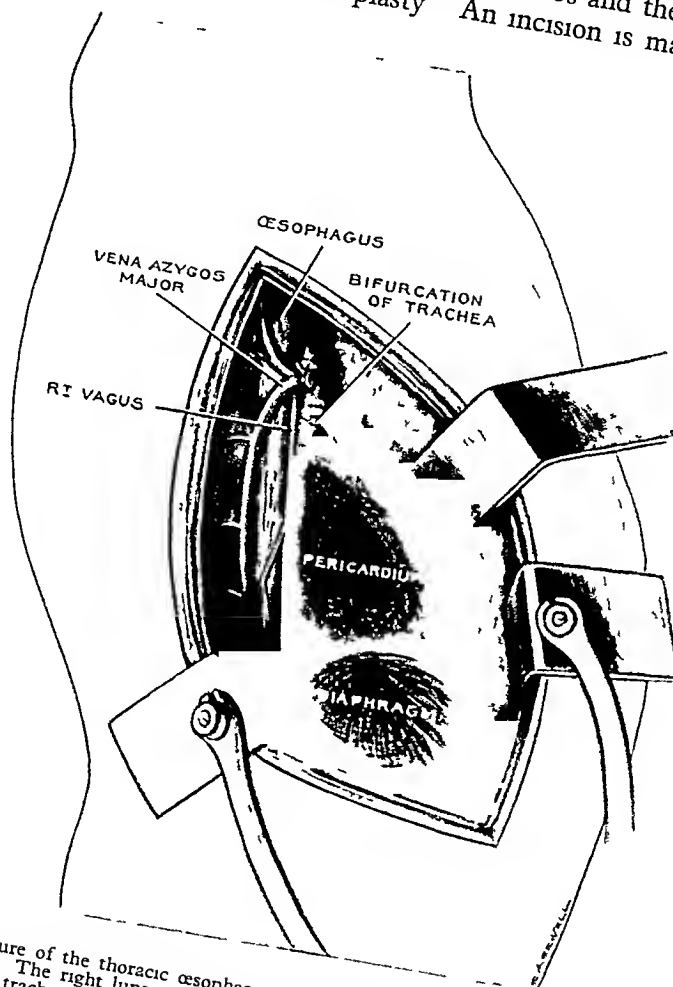


FIG 241—Exposure of the thoracic œsophagus below the bifurcation of the trachea by the right transpleural approach. The right lung is retracted forward and the œsophagus is seen lying between the bifurcation of the trachea, left bronchus, and pericardium anteriorly, and the vena azygos major posteriorly.

commencing at the costal cartilage and extending upwards and backwards over the inferior angle of the scapula. The periosteum covering this rib is elevated and 24 cm of the rib removed. The pleural cavity is opened through the sixth intercostal space. The left lung is retracted forwards and the œsophagus is identified lying between the descending thoracic aorta behind and the pericardium in front. The diaphragm is below, and in the inner part of the wound the termination of the left phrenic nerve with accompanying blood-vessels may be seen. The

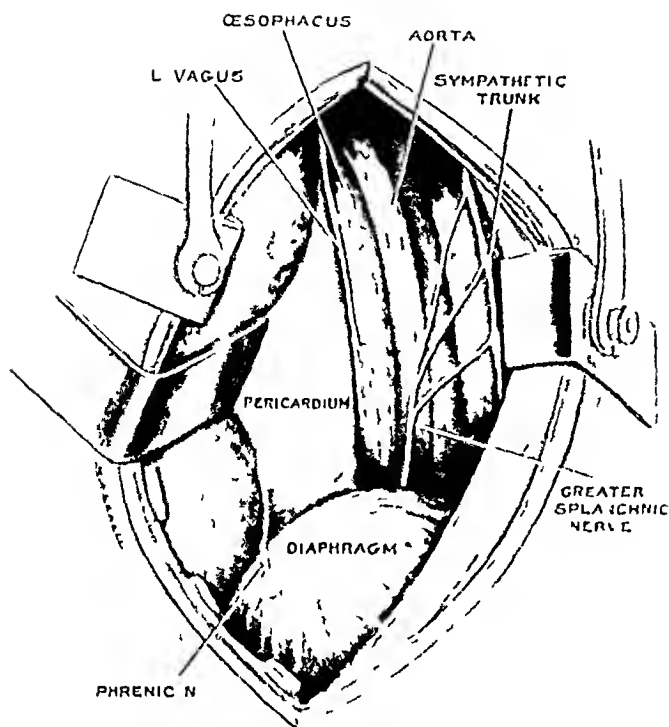


FIG 242 —Exposure of the thoracic œsophagus from the arch of the aorta to the diaphragm by the left transpleural approach. The left lung is retracted forwards and the œsophagus is seen lying between the descending thoracic aorta behind and the pericardium in front. The latter constitutes the greatest obstacle to clear vision of the œsophagus.

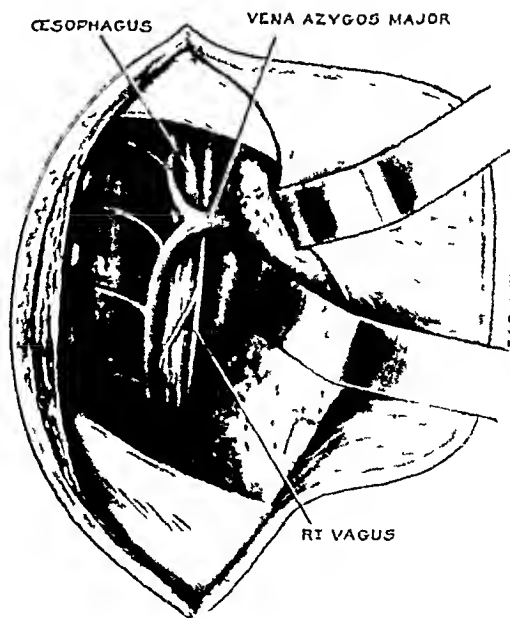


FIG 243 —Exposure of the thoracic œsophagus at the bifurcation of the trachea. The parietal pleura has been separated from the vertebral bodies in a downward direction. The œsophagus is exposed between the separated lung and pleura and the bodies of the vertebrae. The vena azygos major is the important landmark for the œsophagus as it crosses behind the latter to join the superior vena cava.

pericardium constitutes the greatest obstacle to clear vision, but after division of the mediastinal pleura the œsophagus may be drawn outwards and the left vagus with its plexus is easily identified. In this way the œsophagus is exposed between the arch of the aorta and the diaphragm in a length of 14 cm and at a depth of 9.5 cm.

4 *Right Mediastinal Exposure of the Œsophagus (Fig 243)*—The patient lies prone on the table with the right arm held by an assistant as in the operation of paravertebral thoracoplasty. The incision commences 5 cm to the right of the middle line at the level of the second rib and is carried downwards to the angle of the right scapula, at which point it curves outwards to terminate at the level of the eighth rib in the posterior axillary line. After division of the trapezius and rhomboid muscles the scapula is mobilized, as in the operation of thoracoplasty. The periosteum over the fifth, sixth, and seventh ribs, together with the tips of the transverse processes of the corresponding vertebræ, are removed. The lengths of ribs removed are: fifth rib, 7.5 cm; sixth rib, 8.5 cm; seventh rib, 7.5 cm.

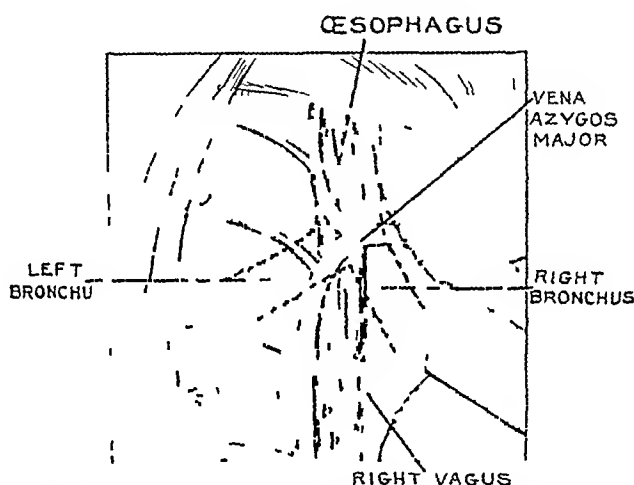


FIG 244—A diagram to show the exact relations of the œsophagus at the level of the bifurcation of the trachea. The œsophagus lies posteriorly to the commencement of the left bronchus and to a lesser extent it comes into contact with the commencement of the right bronchus.

The parietal pleura is gently separated from the vertebral bodies in a downward direction until the œsophagus is exposed lying between the vertebral column and the separated lung and pleura. The lung and pleura are on the right of the œsophagus, and the bodies of the vertebræ are on the left, in front is the trachea and the left bronchus, and at the actual bifurcation of the trachea the commencement of the right bronchus also forms an anterior relation (Fig 244). The vena azygos major passes upwards on the left margin of the œsophagus and at the level of the root of the right lung passes behind the œsophagus to join the superior vena cava. The right vagus nerve is seen on the right of the œsophagus giving a well-marked branch to the posterior aspect. In this way 8 cm of the œsophagus may be exposed at a depth of 6.5 cm.

5 *Left Mediastinal Exposure of the Lower Œsophagus (Fig 245)*—The patient is placed prone on the table with the left hand held above the head by an assistant. The incision commences at the level of the third rib, three finger-breadths below

the mid-line, and extends vertically downwards until it takes an outward sweep along the axis of the eleventh rib. The periosteum covering the posterior aspects of the ribs number four to eleven is exposed and elevated, and 6 cm of each rib is removed. The length removed diminishes towards each extremity of the wound. The parietal pleura is gently separated in a downward direction, commencing near the mid-line. The œsophagus lies between the separated lung and pleura and the descending thoracic aorta. To the inner side of the aorta the bodies of the thoracic

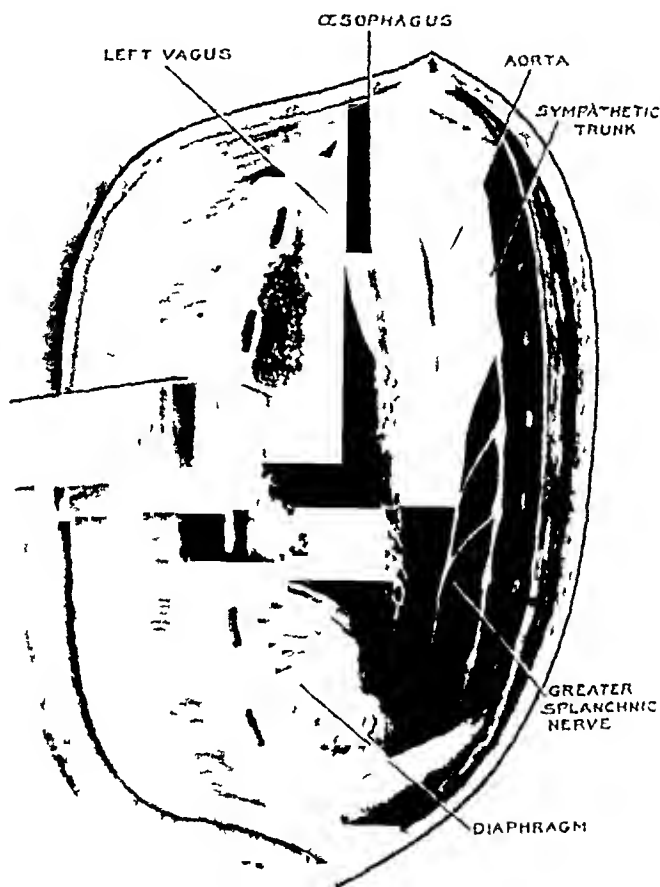


FIG. 245.—Exposure of the thoracic œsophagus from the arch of the aorta to the diaphragm by the left mediastinal approach. The parietal pleura is separated from the vertebral bodies in a downward direction and the œsophagus is exposed between the separated lung and pleura and the descending thoracic aorta.

vertebræ may be seen with the thoracic trunk of the sympathetic and the great splanchnic nerve. The œsophagus disappears above under the arch of the aorta, and, below, it may be seen passing through the œsophageal hiatus of the diaphragm. In this way the œsophagus may be exposed from the arch of the aorta to the diaphragm in a length of some 14 cm. A similar incision but with the sacrifice of fewer ribs permits of a more limited exposure. The œsophagus lies at a depth of 7.5 cm.

THE ABDOMINAL ŒSOPHAGUS

Reference has already been made to the successful operation by Grey Turner in which a carcinoma of the œsophagus was extirpated by the collo-abdominal route. His abdominal approach was made through a median incision, and after separation of the left lobe of the liver from the diaphragm, the œsophagus was exposed.

A carcinoma of the lower œsophagus was excised by Gohrbrandt from below the diaphragm and continuity was restored. The abdomen was opened by an oblique incision below the left costal border. The cardiac end of the stomach was drawn towards the surface, and after section of the left vagus and transverse division of the peritoneum a blunt separation of the lower end of the œsophagus was carried out. Free mobilization was only possible after section of the right vagus. Gohrbrandt recommends that separation should be carried out by the index finger rather than by any special form of instrument, in order to avoid an accidental wound of the pleura as the viscus is followed into the mediastinum. Grey Turner has also used this method. Clairmont has employed this route for the excision of an epiphrenal diverticulum. Grey Turner has made use of the abdominal approach for the operation of œsophagogastrostomy in cases of cardiospasm and simple stricture, and a similar approach has been used by Fromme. Ohsawa has recently reported 14 cases of excision of the lower œsophagus with restoration of continuity, and in 8 of these cases healing resulted. In cases when the exact site of the lesion is doubtful he begins by an abdominal approach. If extirpation by this route seems impracticable, he divides the costal cartilage after the manner of Marwedel and then opens the chest by incising the diaphragm from the costal margin to the œsophageal hiatus.

Technique —

1 *Simple Laparotomy*—The patient lies on his back in the reversed Trendelenburg position—that is, with the foot of the table inclined downwards. A median incision from the ensiform to the umbilicus serves to open the abdominal cavity. After division of the left lateral ligament the left lobe of the liver may be retracted to the right and the region of the cardia exposed. The stomach is drawn downwards and the abdominal œsophagus comes into view. After transverse division of the peritoneum blunt dissection, followed by gentle traction, renders it possible to expose some 5 cm of the œsophagus at a depth of 13 cm.

2 *Marwedel Method (Modified)* (Figs 246–248)—The patient lies in the reversed Trendelenburg position and a vertical incision is made from the root of the ensiform process to a point 2 cm below the umbilicus. At the juncture of the upper two-thirds and lower one-third of this line a second horizontal incision passes out to the costal border (Fig 246). A triangular musculo-cutaneous flap is dissected upward to expose the lower costal cartilages on the left side. After section of the sixth and seventh costal cartilages at their

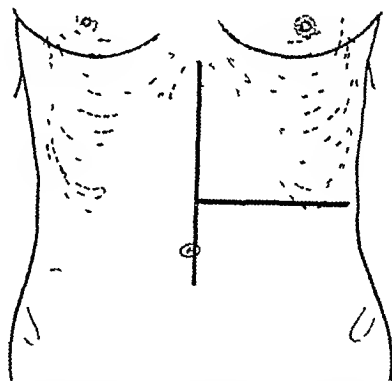


FIG 246—The skin incision for the exposure of the abdominal œsophagus by the Marwedel method

junction with the sternum a subperiosteal resection of some 2 cm of the seventh, eighth, and ninth ribs at the costochondral junction is carried out (*Fig 247*) This manœuvre enables the left costal border to be lifted up in the manner of a trap-door, and after division of the left lateral ligament and retraction of the left lobe of the liver an excellent exposure of the œsophagus is obtained In this way, after division of the peritoneum, mobilization of the œsophagus, and section of both vagi, some 8 cm of the œsophagus may be exposed The original depth from the surface is 14 cm, but mobilization of the organ, together with retraction of the costal flap, enables the œsophagus to be brought up almost to the surface of the wound (*Fig 248*)

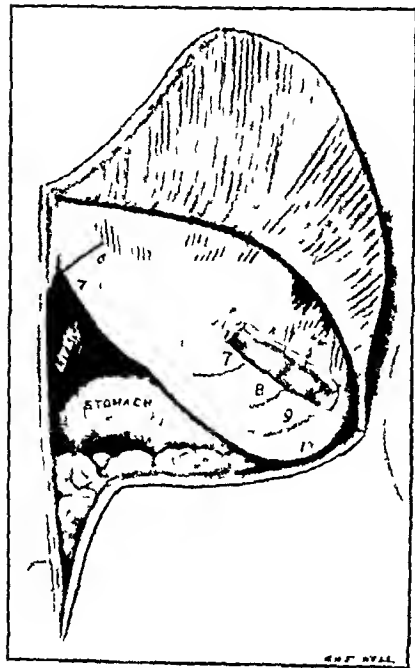


FIG 247—The intervention on the costal margin is the second stage in the exposure of the abdominal œsophagus by the Marwedel method The diagram shows the formation of the hinged flap, which is turned upwards and outwards

CONCLUSION

In approaching the œsophagus by the transpleural route in the dissections, rib resection has always been practised A similar exposure may be obtained by an intercostal incision with removal of segments of the ribs posteriorly, as practised by Lilienthal, or by division of the costal cartilages anteriorly, as

done by Kirschner In a recent operation by one of us (L O'S) the former manœuvre considerably assisted in carrying out a left transpleural exposure of the œsophagus

The upper œsophagus from the level of the thoracic inlet to the lung root is best exposed by a right transpleural approach

The œsophagus in the region of the lung root is best exposed by a right mediastinal approach Excision of short segments of rib renders adequate exposure difficult, but if the lengths described are excised, there is ample room for the assistant's hand to retract lung and pleura

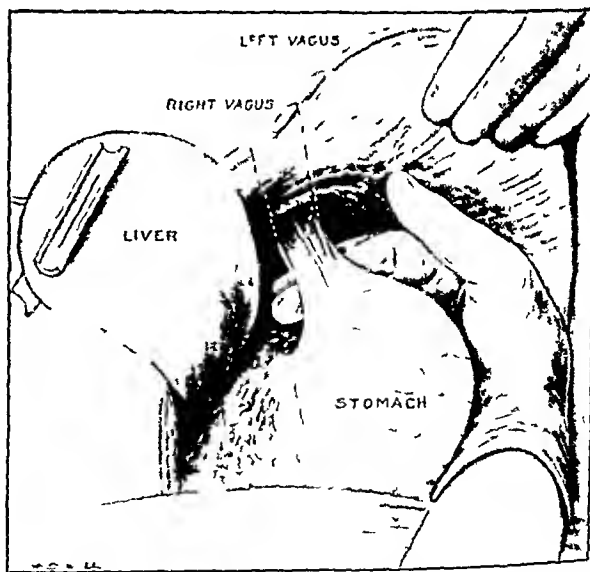


FIG 248—Exposure of the abdominal œsophagus by the Marwedel method The left lobe of the liver has been mobilized to the right and the hinged flap consisting of the lower ribs has been turned upwards and outwards and the abdominal œsophagus is lifted to the surface

without encroachment on the operative field. It is also possible to expose this region by a right transpleural approach after ligature and division of the azygos vein.

The lower œsophagus—from the level of the aorta to the œsophageal hiatus in the diaphragm—is best exposed by a left transpleural approach. If there is any doubt as to the upper limit of the lesion, the right transpleural approach should be substituted, because ligature and division of the vena azygos major enables the organ to be followed upwards, while separation of the œsophagus from behind the arch of the aorta is an intervention of great magnitude. It is also possible to expose the lower œsophagus by the mediastinal route, and here also approach from the left side is to be preferred.

Approaching the œsophagus from below, the modified Marwedel incision seems to be the method of choice if a difficult operation such as local resection with restoration of continuity is contemplated. In suitable cases the operation could be carried out in two stages, with mobilization of the costal arch as the first. Testing the method of Ohsawa on the cadaver, we have found that his method of opening the chest from the abdomen when following the œsophagus up towards the lung root is open to some objections: an incision in the diaphragm stretching from the œsophageal hiatus to the costal border is liable to wound the pericardium, and the closure of this incision offers great technical difficulty. At the same time this method does permit of a preliminary exploration from the abdomen, and although we personally should prefer to begin with the left transpleural approach and then, if access to the cardiac end of the stomach was desired, divide the diaphragm from above—with the pericardium intact—the results obtained by Ohsawa are so remarkable that it seems impossible to dismiss his suggestion lightly.

BIBLIOGRAPHY

- BARRETT, N. R., *Lancet*, 1933, 1, 1009
 CLAIRMONT, P., *Zentralb. f. Chir.*, 1924, 11, 42
 EGGERS, C., *Arch. of Surg.*, 1925, 5, 361
 ENDERLEN, *Deut. Zeits. f. Chir.*, 1901, 141, 441
 EVANS, A., *Brit. Jour. Surg.*, 1933, 10, 388
 FICK, W., *Deut. Zeits. f. Chir.*, 1929, 100, 176
 FROMME, *Arch. f. klin. Chir.*, 1929, 147, 606
 GOHRBRANDT, E., *Ibid.*, 1927, 145, 528
 GREGOIRE, R., *Jour. de Chir.*, 1923, 221, 673
 GREY TURNER, G., *Lancet*, 1933, 11, 1315
 GREY TURNER, G., *Proc. Roy. Soc. Med.*, 1934, Feb., xxvii
 GREY TURNER, G., *New Eng. Jour. Med.*, 1931, 105, 657
 VON HACKER, V., and LOTHEISSEN, G., *Chirurgie der Speiseröhre*, 1926. Stuttgart. Enke
 HEDBLUM, C., *Surg. Gynecol. and Obst.*, 1922, 35, 284
 KIRSCHNER, O., *Arch. f. klin. Chir.*, 1920, 144, 606
 KRAUSS, H., *Med. Klin.*, 1933, 28, 1543 (extr. *Lancet*, 1934, 1, 665)
 KUTTNER, H., *Zentralb. f. Chir.*, 1921, 148, 846
 LEVY, W., *Arch. f. klin. Chir.*, 1922, 146, 20
 LILIENTHAL, H., *Ann. of Surg.*, 1921, 134, 259
 MARWEDEL, G., *Zentralb. f. Chir.*, 1903, 133, 938
 OHSAWA, T., *Arch. f. Jap. Chir.*, 1933, 3, 1
 SAUERBRUCH, F., *Chirurgie der Brustorgane*, 1925, 11. Berlin. Springer
 SCHELBERT, W., *Bruns' Beitr.*, 1914, 10, 410
 SOUTTAR, H. S., *Lancet*, 1933, 11, 285
 TOREK, F., *Deut. Zeits. f. Chir.*, 1913, 103, 305
 WILKIE, D. P. D., and HARTLEY, J. N. J., *Brit. Jour. Surg.*, 1922, 5, 81
 ZAAIJER, J. H., *Bruns' Beitr.*, 1913, 103, 419

THE EXPERIMENTAL PRODUCTION OF CHOLESTEROSIS (STRAWBERRY) GALL-BLADDER

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THE stippled deposits of cholesterol found in the mucous membrane of the gall-bladder in the condition known as strawberry or cholesterosis gall-bladder have excited interest partly because of their possible significance in the etiology of gall-stones, and partly because of their possible relation to clinical symptoms. There are two chief theories on their mode of development. The first, which has the support of Aschoff and Bacmeister, Illingworth, and Berendes, postulates that normally the gall-bladder mucosa absorbs cholesterol from the bile, and that in these cases of cholesterosis gall-bladder there is a breakdown in this absorbing mechanism so that the cholesterol becomes deposited out in the wall of the gall-bladder, various causes for the breakdown, such as chronic inflammation and lymphatic obstruction, have been suggested. On the other hand, there is the theory recently supported by Elman and Graham that the condition results from a breakdown in a normal cholesterol-secreting mechanism in the gall-bladder. The object of the present paper is to present experimental evidence that neither of these theories is necessarily correct, and to discuss the possible clinical importance of this.

LITERATURE

Most of the recorded experimental work has been done on the rabbit, though Picealuga has claimed to have produced the condition in the dog. The dog, however, may normally show deposits of cholesterol in the mucosa of the gall-bladder (Boyd, Chauffard). The gall-bladder of the rabbit, on the other hand, is normally quite free from cholesterol deposits, and most experimental work has been done in this animal. It is well known that if the rabbit is fed with large doses of cholesterol the blood-cholesterol rises and deposits are found in various situations, the most striking being the liver, aorta, suprarenal, and kidney. Though deposits of cholesterol may rarely be found in the gall-bladder under such circumstances, it is generally agreed that they are insignificant in amount. If cholesterol is deposited in the biliary passages at all, it seems to be more frequent in the bile-ducts than in the gall-bladder (Chalatow, Zinslerling). Illingworth has shown, however, that if, in addition to the cholesterol feeding, chronic inflammatory changes are induced in the gall-bladder, a condition histologically comparable in every respect with the cholesterosis gall-bladder of man can be produced. Illingworth produced his inflammatory changes by the direct injection of short-chained streptococci into the gall-bladder wall, and he concluded, partly from histological evidence and partly from chemical evidence, that the deposits of cholesterol resulted from an interference with a normal cholesterol-absorbing mechanism in the gall-bladder.

SCOPE OF PRESENT EXPERIMENTS

Experiments were performed on nineteen rabbits, the general principle of which was that the blood-cholesterol was raised by feeding and at the same time attempts were made to produce varying degrees of inflammatory reaction in the gall-bladder by such procedures as pinching with forceps, puncturing with a hypodermic needle, tying the cystic duct, or injecting organisms intravenously. The animals were given $\frac{1}{2}$ to 1 gram of cholesterol daily for periods up to two and a half months, by which time the blood-cholesterol had risen from the normal region of 100 mgrm per 100 c.c. to the region of 1000 mgrm. Any operative procedure on the gall-bladder was usually carried out before starting the cholesterol feeding. Periodic estimations were made of the blood-cholesterol, and in some cases of the bile-cholesterol. At the conclusion of the experiment the animal was killed, a general post-mortem examination made, and the gall-bladder removed with a piece of adjacent liver and fixed in formalin together with any other organs or tissues to be examined. Frozen sections were subsequently cut, stained with Sudan III, and examined with the polarizing microscope.

Results—A summary of the experimental procedures performed, the main chemical findings, and the condition of the gall-bladder at the conclusion of the experiment is given in the table on p. 380.

THE PATHOLOGICAL AND HISTOLOGICAL FINDINGS

The animals at the post-mortem examination showed the usual infiltrations with cholesterol that are met with in rabbits after prolonged hypercholesterolaemia. Thus the liver was as a rule pale to the naked eye, and grossly infiltrated with cholesterol microscopically, and the characteristic deposits were present in the aorta often in marked degree. In addition, the region of the incision in the abdominal wall was usually pale like the liver, and microscopical examination confirmed that it too was grossly infiltrated with cholesterol, chiefly in the areas of inflammatory cell infiltration (*Figs 249, 250*).

The appearance of the gall-bladder varied. In some of the animals no inflammatory changes were produced in the gall-bladder by the experimental procedure employed. Such was the case, for example, in the animals injected intravenously with short-chained streptococci. In these the gall-bladder was normal on both naked-eye and microscopical examination, and also devoid of cholesterol deposits. In other experiments, in which the external injury to the gall-bladder had been slight, there was inflammatory thickening of the serosa, characterized by fibrosis and some mononuclear proliferation, while the other coats were practically free of inflammatory changes. In these cases there were deposits of cholesterol in the inflamed areas in the serosa, but not in the rest of the gall-bladder (*Figs 251, 252*).

On the other hand, in the animals in which the injury to the gall-bladder had been severe, there were fibrosis and gross inflammatory changes throughout the whole of the gall-bladder, often accompanied by sloughing and disappearance of the epithelial lining, and deposits of cholesterol could be seen microscopically scattered throughout the whole thickened gall-bladder. Often the gall-bladder was surrounded by adhesions or buried by overlapping lobes of the liver, and cholesterol

SUMMARY OF EXPERIMENTS

Exp No	OPERATIVE PROCEDURE	BLOOD-CHOLESTEROL AT START OF EXP	BLOOD-CHOLESTEROL HIGHEST REACHED	BILE-CHOLESTEROL AT START OF EXP	BILE-CHOLESTEROL HIGHEST REACHED	CONDITION OF GALL BLADDER POST MORTEM	CHOLESTEROL DEPOSITS IN GALL BLADDER
		Mgram per 100 c c	Mgram per 100 c c	Mgram per 100 c c			
1	Injection of olive oil into G B	105	875	172 *	415	Normal	Absent
2	Pinching G B with forceps	115	1330	157 *	250	Thickened and opaque	Present chiefly in subserosa
3	As 2	100	910	137 *	490	Thickened and opaque	Present slight in amount
4	Repeated aspiration of G B	125	924	55	150	Normal	Absent
5	As 4	195	860	45	200	Buried in adhesions	Present in adhesions not in G B
6	As 4	145	1300	50	340	Normal	Absent
7	As 4	88	1220	—	250	Post-mortem decomposition present and not examined microscopically	
8	As 4	94	1350	120	170	Thickened and opaque	Not microscopied
9	As 4	120	1030	—	200	Thickened and opaque	Well marked deposits
10	Pinching with forceps and tying cystic ducts	150	1140	—	—	Normal	Absent
11	As 10	150	960	—	—	Markedly thickened and inflamed	Present
12	As 10	150	900	—	—	Normal	Absent
13	As 10	105	810	—	—	Slightly thickened	Absent
14	Intravenous injection of streptococci from case of gall-stones	145	790	—	—	Normal	Absent
15	As 14	90	790	—	—	Normal	Absent
16	As 14	120	690	—	—	Markedly thickened and inflamed	Present in slight amounts
17	Cystic duct tied, G B pinched with forceps	—	—	—	—	Slightly inflamed	Present
18	As 17	—	685	—	—	Markedly thickened	Present
19	As 17	—	1000	—	—		

* After 17 days feeding

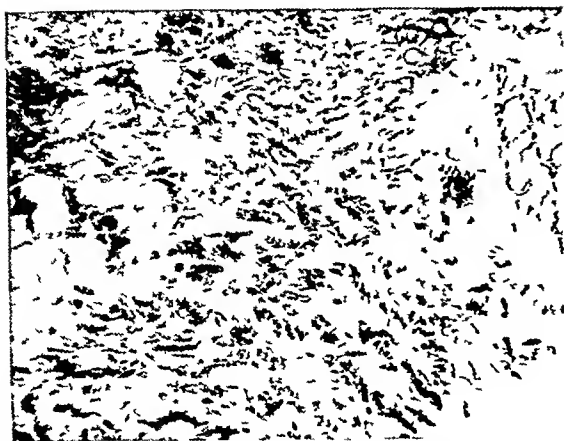


FIG 249—Section of anterior abdominal wall in the region of the incision, stained with hæmatoxylin and Sudan III, and showing collections of granular chronic inflammatory cells interspersed between the muscle fibres ($\times 45$)

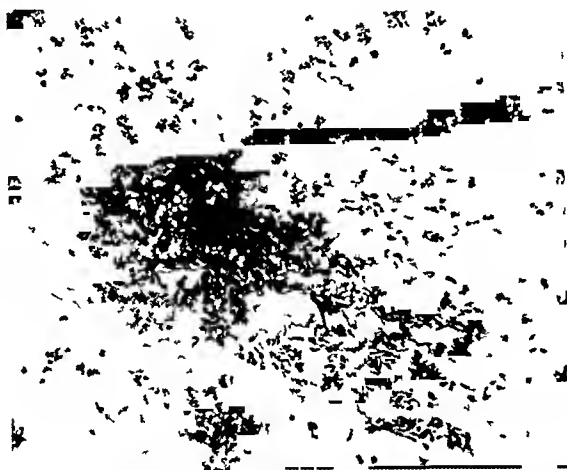


FIG 250—Part of the same section as Fig 249 examined with the polarizing microscope, and showing the doubly refractile cholesterol ($\times 65$)



FIG 251—Section of gall-bladder stained with hæmatoxylin and Sudan III, showing above the villi of the mucosa of the gall-bladder, and below the grossly thickened serous coat containing numerous granular inflammatory cells ($\times 45$)

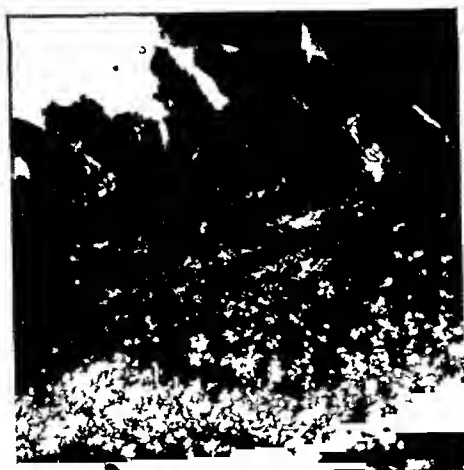


FIG 252—Part of the same section as Fig 251 examined with the polarizing microscope, and showing the doubly refractile cholesterol confined to the thickened serous coat ($\times 65$)

could be demonstrated in the adhesions. But in a few of the animals the inflammatory changes were most marked towards the epithelial surface, accompanied by deposits of cholesterol in the villi of the mucosa (*Figs 253, 254*). A picture was thus produced exactly comparable with that found in the strawberry gall-bladder of man. The cholesterol deposits stained with Sudan III, and were

FIG 253—Section of gall bladder stained with hematoxylin and Sudan III, and showing above the epithelial surface and two villous folds, and below the adjacent liver. The villous folds are infiltrated with chronic inflammatory cells ($\times 50$)



doubly refractile on examination with the polarizing microscope, and on examination with the high power seemed to be chiefly present in phagocytic cells, as in *Fig 255*, which shows similar cells from the anterior abdominal wall. The presence of cholesterol in the gall-bladder thus depended on whether inflammatory changes were present, and their situation. It is worthy of note, however,



FIG 254—Part of the same section as *Fig 253* examined with the polarizing microscope, and showing the doubly refractile cholesterol in the stroma of the villi ($\times 75$)

that the amount of cholesterol deposited was not directly proportionate to the amount of inflammatory reaction, and in the grossly thickened fibrotic gall-bladders the deposits were relatively scanty.

The contents of the gall-bladder in the cases in which only minor trauma had been inflicted consisted of apparently unaltered bile, but in the cases in which gross trauma such as pinching with artery forceps had been inflicted,

blood-clot in various stages of inspissation up to that of hard black concretions was found. There was, however, in no case anything in the gall-bladder comparable to human gall-stones.

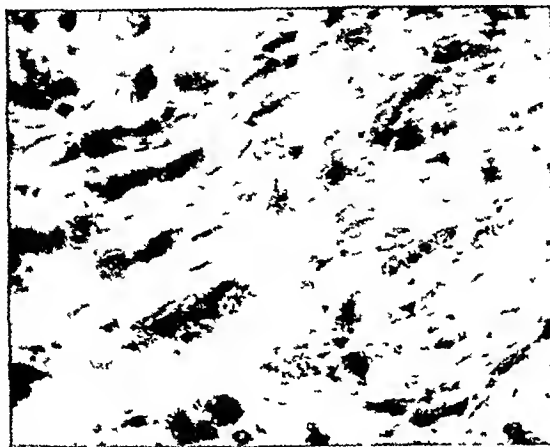


FIG 255 —High-power view of the same field as Fig. 249, showing the granular lipoid-containing cells ($\times 200$)

THE CHEMICAL FINDINGS

In those experiments in which estimations were made of the cholesterol in the bile aspirated from the gall-bladder as well as of the blood-cholesterol, the level of the cholesterol in the bile was always found to be much less than in the blood. Thus in one animal (Experiment 6) the blood-cholesterol rose to 1300 mgrm per 100 c c, while the bile-cholesterol was only 340, and in another (Experiment 8) in which the blood-cholesterol rose to 1350, the bile was only 170.

The question whether there is a direct relation between the level of the cholesterol in the blood and in the bile is in dispute, and while some theories of gall-stone formation are based on the view that there is such a direct relation, others deny that raising the blood-cholesterol has any influence at all on the bile-cholesterol (Dostal and Andrews). Most of the present experiments are not capable of being brought to bear on this problem, because in most of them relatively gross trauma was deliberately inflicted on the gall-bladder, and it is certain that some of the rise in the bile-cholesterol must have been due to this. But in some of the experiments the possibility that raising the blood-cholesterol raises the bile-cholesterol cannot be dismissed. For example, in Experiment 6 no injury was done to the gall-bladder other than that associated with pricking it with a fine hypodermic needle to aspirate the bile for the estimation. At the start of the experiment the cholesterol reading was 50 mgrm per 100 c c of bile, at the end of fifty-seven days' feeding, when the blood-cholesterol was 1300, the bile-cholesterol was 340, and on the cholesterol feeding being omitted, the bile-cholesterol fell to 113 and subsequently to 100. In another similar experiment (Experiment 8) the bile-cholesterol rose from 120 to 170 with cholesterol feeding, and fell to 110 on its omission.

While it must be admitted that the rabbit is not a very satisfactory animal for this type of experiment owing to the small size of the gall-bladder, the above results suggest that a raising of the blood-cholesterol is accompanied by a raising of the bile-cholesterol, but small in degree in comparison with that of the blood

DISCUSSION

The present experiments thus confirm those of Illingworth in showing that it is quite easy to produce a typical strawberry gall-bladder in rabbits by a combination of cholesterol feeding and the production of chronic inflammatory changes. The evidence, however, is against Illingworth's conclusion that the cholesterol comes from the bile and is deposited in the gall-bladder wall owing to a breakdown in a normal absorptive mechanism, and the following summarizes the evidence against this conclusion—

1 In those animals in which the inflammatory reaction was confined to the serosa, the deposits of cholesterol too were confined to the serosa

2 Similar deposits of cholesterol were present in other chronically inflamed areas—for example, the incision in the abdominal wall and the adhesions around the gall-bladder

3 Moreover, tying the cystic duct before the cholesterol feeding started did not prevent deposits of cholesterol in the inflamed gall-bladder (Experiments 11, 13, 17, 18, 19)

4 Finally, there is the chemical evidence, which, so far as it goes, suggests the blood rather than the bile as the source of the deposits of cholesterol in the gall-bladder, in that the blood figures were always so much higher than those of the bile

The evidence against the view that the experimentally produced cholesterosis gall-bladder of rabbits results from a breakdown in a normal cholesterol-absorbing mechanism thus seems conclusive. There is equally little justification for postulating a breakdown in a normal cholesterol-secreting mechanism, and the obvious explanation is that a chronically inflamed gall-bladder in a hypercholesterolized rabbit is liable to have cholesterol deposited in it from the blood in the same way as other chronically inflamed areas. The cholesterol is probably carried by wandering phagocytic cells (Zinscring), and, as already noted, such lipid-containing cells can in most cases be clearly demonstrated (*see Fig 255*)

APPLICATION OF EXPERIMENTAL FINDINGS TO MAN

Admitting that typical strawberry gall-bladder can be produced in the rabbit and that the cholesterol is deposited from the blood in phagocytic cells, the question arises of the application of the experimental findings to man. The metabolism of cholesterol in herbivorous animals is so different from that in carnivorous animals that very great caution must be exercised before applying conclusions derived from one to the other. In man cholesterosis gall-bladder occurs without the high blood-cholesterol which is necessary in the rabbit, and usually without gross inflammatory changes in the gall-bladder, so that in the present case caution is particularly necessary. All that can be claimed, therefore, from the present experiments is merely that they provide an alternative possibility to the usual theories of the mode

of development of cholesterosis gall-bladder, the proof or refutation of this possibility depending on investigations in man

The question whether the condition represents a depositing of cholesterol out of the blood similar to that which occurs in other xanthomatous areas is not merely of academic interest, but of practical importance in treatment. If the general view of the etiology is correct, a marked cholesterosis gall-bladder indicates an organ one function of which is so seriously impaired that the question of removal arises as a rational form of treatment. And since the condition cannot usually be diagnosed from external inspection alone, the surgeon might feel called upon when operating on a case in which the symptoms were equivocal to remove a gall-bladder which appeared normal externally, in order to be quite sure he was not leaving behind such a functionally abnormal organ. And, in actual fact, the demonstration of these deposits in an otherwise healthy gall-bladder that has been removed by operation is commonly regarded by surgeons as a pathological justification for the operation of cholecystectomy. If, however, cholesterosis gall-bladder represents merely a depositing of cholesterol out of the blood, then, provided the functional tests are satisfactory and the organ appears otherwise healthy, there would seem to be no more indication for removal of the gall-bladder for the condition *per se* than there is for the removal of other xanthomatous areas.

Illingworth has described cases of cholesterosis gall-bladder associated with symptoms referable to the gall-bladder which were cured by cholecystectomy. The tests of gall-bladder function were normal in these cases. It is difficult to see in what way deposits of cholesterol in the mucosa of an otherwise healthy and normally functioning gall-bladder could cause symptoms, and it seems certain from the figures which Berendes gives on the high incidence of the condition in routine post-mortem examinations that in the majority of cases cholesterosis gall-bladder is unassociated with symptoms. Further clinical investigations on the lines of those of Illingworth would therefore be of great interest, and might throw light on a point of considerable importance in gall-bladder surgery—that is, whether it is justifiable to remove a gall-bladder on symptoms alone in the absence of calculi or other external evidence of gall-bladder disease.

The present investigation provides no evidence on the relation, if any, between cholesterosis gall-bladder and gall-stones, and, owing to the differences between animals and man in the composition of the bile and in metabolism generally, it is likely that this question will only be solved in man. That the two conditions are causally related is suggested by numerous clinical cases in which cholesterol gall-stones have been found associated with cholesterosis gall-bladder, and in which it is very tempting to assume the sequence of diffuse cholesterosis, cholesterol polyposis, and finally cholesterol gall-stones. On the other hand, it has already been noted that it is only in a minority of cases that cholesterosis is associated with gall-stones (Berendes), and the possibility that the simultaneous presence of the two conditions is coincidental cannot be dismissed. Thus in a gall-bladder recently removed from a middle-aged woman, both diffuse cholesterosis and cholesterol polyposis of the gall-bladder were associated with calculi composed predominantly of pigment and calcium, and containing only 30 per cent cholesterol, suggesting that in this case at any rate the two conditions were merely coincidental. In the present state of our knowledge, therefore, the relation between cholesterosis

gall-bladder and gall-stones is too obscure to justify any practical conclusions, and certainly too obscure to justify anything in the nature of prophylactic removal of the gall-bladder.

SUMMARY

1 Experiments are described confirming those of Illingworth in showing that in the rabbit cholesterosis gall-bladder similar histologically to that of man can be produced by a combination of raising the blood-cholesterol and inducing chronic inflammatory changes in the gall-bladder

2 It is concluded that the experimental condition arises by a direct depositing of cholesterol from the blood in phagocytic cells similar to that which occurs in other inflamed areas, rather than through an interference with a cholesterol-absorbing mechanism

3 The practical importance of this is discussed, and its possible bearing on the surgery of the gall-bladder

This work was carried out by me in the Bland-Sutton Institute of Pathology of the Middlesex Hospital as Streatfield Research Scholar of the Royal College of Physicians of London and the Royal College of Surgeons of England, and I have much pleasure in acknowledging my indebtedness to Professor James McIntosh, Director of the Bland-Sutton Institute of Pathology, for providing me with facilities for carrying out the work and for his kindness and help. I also wish to thank Professor E. C. Dodds, Director of the Courtauld Institute of Biochemistry, for his kind help with the chemical estimations

BIBLIOGRAPHY

- ANITSCHKOW, N, *Arch f Dermatol*, 1914, cxx, 627
 ASCHOFF, L., and BACMEISTER, A, *Die Cholelithiasis*, 1909 Jena Fischer
 BERENDES, J, *Arch f klin Chir*, 1933, clxxv, 266
 BOYD, W, *Brit Jour Surg*, 1923, x, 337
 CHALATOW, S. S., *Beitr z pathol Anat*, 1914, lvii, 85
 CHAUFFARD, *La Lithase biliaire*, 1922 Paris Masson
 DOSTAL, L. E., and ANDREWS, E., *Arch of Surg*, 1933, lxxvi, 258
 ELMAN, R., and GRAHAM, E. A., *Ibid*, 1932, lxxiv, 14
 ILLINGWORTH, C. F. W., *Brit Jour Surg*, 1929, lvii, 203
 PICCALUGA, N., *Arch ital di chir*, 1927, x, 125
 ZINSERLING, W., *Beitr z pathol Anat*, 1923, lxxi, 292

SHORT NOTES OF RARE OR OBSCURE CASES

PRIMARY CARCINOMA OF THE LIVER: SUCCESSFUL LOBECTOMY

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PRIMARY carcinoma of the liver is an uncommon condition, and a victim is very fortunate when the disease is discovered while still localized to the left lobe, and so lends itself to removal

HISTORY AND EXAMINATION—W M, male, aged 59 Three years ago while lifting a heavy weight, the patient suddenly felt a 'tearing' sensation in the pit of the stomach Two days later he felt faint and vomited a considerable amount of blood, estimated at two pints He was admitted to hospital and a bismuth meal showed no evidence of gastric abnormality His condition improved and he returned to work, but found that any effort made while in a stooping position caused epigastric discomfort Four months ago continuous abdominal pain supervened which bore no relationship to food, rest, or exercise, and the patient noticed that he was losing energy and becoming progressively less fit for work

On Feb 6, 1934, the patient discovered an epigastric swelling which led him to consult Dr C W Seccombe, of Southall, who diagnosed hepatic enlargement, and recommended further investigation The patient was accordingly admitted to hospital, and presented himself with an obvious swelling in the left epigastrium, which was the size of a coconut The tumour was definitely tender, firm in consistency, and evinced transmitted aortic pulsation, which was less conspicuous in the genu-pectoral position A blood examination showed no abnormality beyond a slight leucocytosis, and pancreatic tests were normal A bismuth meal indicated the presence of a solid swelling between the stomach and the left cupola of the diaphragm It was decided that a laparotomy should be performed

OPERATION (Feb 27)—General anæsthesia The abdomen was opened through a left upper paramedial incision The left lobe of the liver presented itself as a rounded mass, slightly irregular on the surface, obviously congested, and of elastic consistency An exploring needle withdrew blood It was then recognized that the tumour was in all probability carcinomatous, and the intestinal tract was carefully searched for a primary growth The examination was entirely negative, the right lobe of the liver appeared to be normal, and the peritoneal cavity contained no free fluid It was then decided that the hepatic tumour was probably a primary carcinoma, and as the patient's condition was satisfactory an attempt to remove the left lobe was justifiable In order to gain adequate exposure the rectus muscle and parietes were divided in an upward and outward direction from the level of the umbilicus to the costal margin The falciform and left coronary

ligaments were rapidly divided, which permitted the left lobe to be drawn to the right so as to expose the portal fissure. Brief dissection revealed the left hepatic artery and the left branch of the portal vein, which were secured with clamps. A few interlocking sutures were inserted into the isthmus of liver substance connecting the two lobes, and the remainder of the liver tissue was divided with an electro-cautery (*Fig 256*). After removal of the left lobe the left hepatic artery and portal



FIG 256 —A section through the left lobe of the liver. The left branch of the portal vein, the left hepatic artery, and the left hepatic duct are seen immediately in front of the charred surface, which was divided with an electro-cautery knife.

vein were ligatured. Haemorrhage from the raw surface of the liver was insignificant. A drainage tube was inserted and the wound closed. A pint of intravenous saline was administered towards the end of the operation, and 250 c.c. of blood were transfused two hours later.

Convalescence was somewhat disturbed by ileus (which responded to pitressin), and haematemesis, which persisted for four days. The haematemesis was presumably due to congestion of the gastric veins following ligation of the left branch of the portal vein. The patient was discharged to a convalescent home sixteen days after the operation.

PATHOLOGICAL REPORT (*Fig 257*)—On microscopic section the tumour was found to be composed of irregularly arranged cuboidal and spheroidal cells, and was considered to be a hepatoma arising from the liver parenchyma. The normal liver tissue showed no evidence of cirrhosis.

SUBSEQUENT HISTORY—Although the patient's condition improved at the convalescent home, he failed to regain his normal strength, and after two weeks subcutaneous nodules appeared in the region of the umbilicus. On section these



FIG 257—Microscopic section of the tumour ($\times 200$)

proved to be identical with the liver growth, and were presumably secondary deposits which had spread along the lymphatics in the falciform ligament. The condition of the patient steadily deteriorated and he died on April 16.

POST-MORTEM EXAMINATION—At necropsy the right lobe of the liver was found to be enlarged and studded with secondary growths. The left lobe was absent and the scar of its removal was clean. There were a few small secondaries in the peritoneum overlying the right kidney, and also in the left hilar glands. One small nodule was present in the heart. There was no lesion in the stomach, duodenum, small intestine, or colon, and the case appeared to be one of primary carcinoma of the liver.

I am indebted to Mr L. Fatt, F.R.C.S., of the Hillingdon County Hospital, for the subsequent history of this case.

A DERMOID CYST OF THE GREAT OMENTUM WITH UNUSUAL ATTACHMENTS

By A L D'ABREU

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CYSTS of the peritoneum are rare of this group omental cysts are the least frequent, and are of five types (1) Lymphatic or chylous, (2) Enterogenous (deriving from sequestered congenital intestinal diverticula or Meckelian remnants), (3) Urogenital cysts, usually of nephrogenic origin, (4) Dermoids and teratomata, (5) Hydatid cysts. The following case is a dermoid cyst of the omentum —

A married female, aged 60, a diabetic, was admitted to the Cardiff Royal Infirmary on the second day of a typical attack of acute appendicitis. Abdominal examination revealed, in addition, an extremely mobile tumour, the size of a golf ball, which could be felt in the left hypogastrium. The swelling was painless, uniformly smooth, and could be pushed with ease in all directions. On vaginal examination the tumour appeared to be unconnected with the pelvic organs. The patient was unaware that her abdomen contained a lump, and there had been no symptoms arising from it.



FIG 258 —Microscopic appearance of the tumour



FIG 259 —Cut surface of the cyst

OPERATION—After insulin had been administered, immediate operation for appendicitis was carried out through a right paramedian incision. An acutely inflamed appendix was removed. I then passed a hand into the abdomen and delivered into the wound a soft spherical mass enmeshed in the great omentum and attached by dense fibrous strands to the anterior abdominal wall in the region of the umbilicus, to the mesenteric border of the ileum about three feet from the ileocaecal valve, and also to the apex of the bladder. There were no connections with the internal genitalia, which were seen to be normal. The swelling with some adherent omentum was removed without difficulty and the abdomen closed. Convalescence was uneventful.

PATHOLOGY—*Fig 258* illustrates the macroscopic appearance of the tumour and its intimate relation to the omentum. To the left of the picture a strand of silk is tied to the fibrous band that ran from the tumour to the ileum. In view of the attachments the diagnosis of vitello-intestinal cyst was made, a urachal origin was also considered in view of the bladder attachments seen at operation. On section, however, the tumour was found to be a typical dermoid cyst containing sebaceous material and hair. *Fig 259* shows the cut surface of the cyst.

COMMENTARY

Before operation the mobility of the tumour suggested a mesenteric cyst, but the ease with which the tumour could be manipulated in a vertical as well as a horizontal axis discounted this diagnosis. There are few recorded omental dermoid cysts. Mantel¹ (1895) described a dermoid cyst of the great omentum. Meckel² (1815) writes, "Ruysch found in a woman who had long suffered from dropsy, a tumour the size of a fist, filled with a white pulpy mass and locks of tangled hair, situated in the omentum which was everywhere as thick as a finger, and firmly adherent to the peritoneum". F Niosi,³ in an article on mesenteric cysts, described a large symptomless lump removed from the omentum which was teratomatous in nature, containing epithelial formations, suprarenal tissue, and an area of chorio-epithelioma in addition to multiple small cysts. This tumour was attached to the mesocolon and was probably of mesenteric rather than omental origin. D P D Wilkie⁴ recorded a case of intestinal obstruction in a boy of 15 due to an omphalo-mesenteric cyst in which the attachments closely resembled those in the case reported above, but the presence of glairy mucoid fluid in the cyst proved the tumour to be enterogenous in origin. Stanford Cade (quoted by Arthur Evans⁵) removed an omental cyst, but microscopical examination showed a lining of columnar epithelium lying on muscularis mucosæ, and this led Evans to classify the case as a developmental enterogenous cyst.

The origin of omental dermoids is obscure. Wilms⁶ believed them to be due to imperfect closure of the abdominal plates, other observers postulate an ovarian source, by development from a supernumerary ovary, from a free ovum, or from a ruptured ovarian dermoid. Such hypotheses remain unsubstantiated. The origin in the case recorded may well be from omphalo-mesenteric or allantoic remnants in view of the attachments present.

I am grateful to Professor A W Sheen, Director of the Surgical Unit, for his help and advice and for permission to publish this case, also to Mr Arthur Evans whose work on enterogenous cysts was so helpful and who took an interest in this case.

REFERENCES

- ¹ MANTEL, K, "Ueber Dermoidkysten der Ovariums", Inaug. Diss. Heidelberg, 1892, quoted by M Wilms, *Deut. Arch. f. klin. Med.*, 1895, lv, 289, 396.
- ² RUYSCH, F, "Observationum Anatomico-chirurgicarum centuria", Amsterdam Obs. 18, Quoted by J E Meckel, *Deut. f. d. Physiol.*, 1815, i, 519.
- ³ NIOSI, F, "Die Mesenterialzysten embryonalen Ursprungs nebst einigen Bemerkungen zur Entwicklungsgeschichte der Nebennieren-Rindensubstanz sowie zur Frage des Chorion-epithelioms", *Virchow's Arch.*, 1907, cxc, 217.
- ⁴ WILKIE, D P D, "Omphalo-mesenteric Cyst causing Acute Intestinal Obstruction", *Brit. Jour. Surg.*, 1915, iii, 145.
- ⁵ EVANS, A, "Developmental Enterogenous Cysts and Diverticula", *Ibid.*, 1929, xvii, 73.
- ⁶ WILMS, Quoted by Ewing, *Neoplastic Diseases*, 1922, 976.

PROGRESSIVE POST-OPERATIVE CUTANEOUS GANGRENE

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In the *Annals of Surgery*, December, 1932, Patterson describes under the heading of "Progressive Post-operative Cutaneous Gangrene", a case of extensive streptococcal ulceration of the skin, with recovery. On searching the literature he was able to collect only twenty similar cases, and, with this small number recorded, one is justified in assuming that the condition must be rare, as such characteristic and remarkable cases would, when met, probably call for publication more frequently.

All the cases so far recorded follow each other closely in detail, and the description of their appearance is so similar that it seems possible to class them as a definite clinical entity. As the title suggests, the condition is a slow but progressive ulceration of the skin in the neighbourhood of a recent operation wound. This ulceration, of characteristic appearance, continues to extend remorselessly in spite of all conservative treatment, and is due to an unusual streptococcal infection belonging to the anaerobic class. The majority of cases, if not all, appear to develop in the puncture wound of a deep tension suture following operations for empyema, perforated acute appendicitis, and perforated duodenal ulcer, and it seems reasonable to suggest that this unusual type of infection originates from within the bowel or lung.

A search of the English literature, which has been kindly carried out by the Librarians of the Royal Society of Medicine, has failed to discover any reference to a similar case, with the only exception in *The Medical Journal of Australia*, 1930.¹ Here under the heading of "Spreading Gangrenous Inflammation" a case is recorded of apparently similar infection, arising in an empyema wound, in which the ulceration ultimately extended over the whole of the back, from neck to buttock, and death followed. For these reasons it is thought that the following recent case should be recorded.

Male, aged 57 years. Previous health good, except for indefinite symptoms of dyspepsia with no characteristic sequence. On June 5, 1933, he suffered from severe abdominal pain, diagnosed as perforated duodenal ulcer, and laparotomy was performed within six hours of onset. A perforated anterior chronic ulcer in the first part of the duodenum was found. This was closed by one purse-string suture and two tags of omentum were stitched over the repair to assist. There was very little soiling of the peritoneum, and the abdomen was closed in layers with continuous catgut sutures and three deep tension sutures of silkworm gut were used, each passing through skin and rectus muscle on both sides of the incision. No drainage was employed. The post-operative period was uneventful. The tension sutures were removed on the eighth day. About the tenth day a bead of pus escaped from the right puncture wound of the upper tension suture and also from the left wound of the lowest tension suture. These two small puncture wounds became more inflamed, ulcerated, and slowly spread very definitely laterally, with little tendency to extend in the long axis of the body, thus leading to two

separate ulcers, one on either side of the incision. There was no encroachment of this process towards the mid-line, and the original incision remained firmly healed throughout with a small margin of healthy skin on each side between it and the spreading ulcers. The spread of the ulcers was very striking. The advancing edge of each ulcer was similar, and throughout tended to spread outwards and in no other direction. The process was preceded by a wide bluish inflammatory zone about 2 to 3 cm wide, and immediately internal to this was a narrow rim of dry gangrenous tissue less than 0.5 cm wide. As this advanced it left behind a thick grey slough which was firmly adherent, and this only slowly separated some ten days later, leaving a clean healthy granulating surface. From beneath this slough there was a constant discharge of pus, and many beads could be expressed by gentle pressure.

The later stages were remarkable. The outer edge of each ulcer was inflamed and advancing, while the old original area near the mid-line was clean and healthy and actually epithelizing over. The involved area was exquisitely tender throughout, and the temperature ranged between 99° and 103°, though apart from this there was remarkably little toxæmia. The whole inflammatory process was entirely confined to the skin and subcutaneous tissue, the abdominal muscles being unaffected and remaining free from infection and functioning normally throughout. The rate of spread was slow but steady. At the end of six weeks each ulcer was about 6 by 3 in., with a bridge of normal skin containing the healed incision separating the ulcers. This gives an average daily advance laterally of approximately 4 mm, while very little extension upwards or downwards had occurred. Cultures from the pus had grown a scanty growth of streptococci, and for this reason treatment by excision had been rejected for fear of opening fresh planes of tissue and disseminating the infection. During this period many different lines of treatment had been employed, including serum, Carrel-Dakin, and other antiseptics, blood transfusion, sodium nucleinate, ultra-violet, and even infra-red, rays. All lines of local and general treatment were singularly conspicuous for their complete failure to allay, even in the slightest degree, the steady progress of the condition.

At this stage Patterson's case was brought to notice, and it was decided to excise completely the inflammatory area forming the advancing edge. This was done on July 21, 1933. The excision was made wide in healthy tissue outside the inflammatory area and carried internally until the region of healthy granulating tissue was reached, thus completely excising the advancing edge of each ulcer down to, but not exposing, the muscles. A large raw area was left after this extensive excision, and two silkworm sutures, one on either side, were inserted to endeavour to reduce this by drawing the skin edges lightly together. There was no untoward general reaction following this, but seven days later all four suture puncture wounds were infected in the same way and showed gangrenous patches round each wound, and pus could be expressed from the deeper surfaces of each. The rest of the wound, however, was extremely healthy. These four points were excised wide of the inflammatory process and no sutures were used again. On two further occasions the extreme edge of the wound developed an area of gangrene and each required excision, the first ten days later, and the second six days following that. The last excision was on Aug. 16. From then onwards the healing was rapid, epithelium growing in with remarkable rapidity. After the last excision an enormous area of skin had been removed, and in view of the previous experience with the use of stitches it was not possible to draw the skin edges together. It was estimated that

well over fifty square inches of skin had been removed, yet within six weeks the whole of this, except for a minute area, had epithelized over and the patient was home, the only dressing used being flavine and paraffin for the first ten days or so, and then a simple saline dressing. *Fig 260* shows the condition of the ulcers immediately prior to the first operation, and *Fig 261* shows the final result

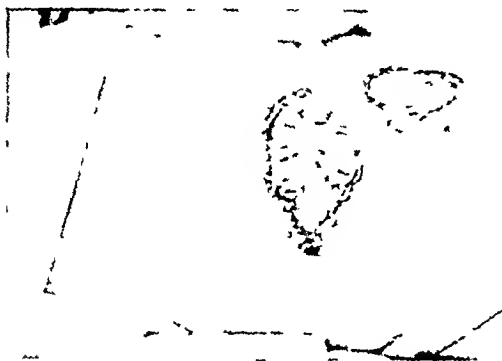


FIG 260—Showing the two ulcers with the gangrenous edge and the healed incision immediately before the first operation for excision



FIG 261—Showing the narrow scar resulting from the very wide excision of skin

BACTERIOLOGICAL REPORT (Dr Facey)—Examination of a piece of tissue from the wound gave the following result. On direct examination two distinct types of Gram-positive cocci were seen, one having the appearance of a streptococcus and the other a staphylococcus. Primary cultures on blood-agar gave no growth aerobically, but a growth of streptococci and staphylococci anaerobically. No growth occurred in broth. On sub-culture both organisms grew aerobically, though the streptococci grew much more profusely under anaerobic conditions. 5 c.c. of a strong emulsion of a twenty-four-hours culture of the streptococci proved non-pathogenic when injected into the peritoneal cavity of a guinea-pig.

SUMMARY

- 1 A case is described of progressive post-operative gangrenous ulceration, of which only about twenty cases have been recorded, mainly in America.
- 2 This ulceration is of characteristic appearance, and all recorded cases resemble each other very closely. The progress of the ulcer is slow but steady, until, if left, an enormous area may be involved and death follows.
- 3 No treatment appears to be of any avail in checking the inflammatory spread except wide excision of the advancing edges of the ulcer and surrounding discoloured area. This can confidently be carried out without fear of disseminating the infection, and is successful in curing the condition.
- 4 The infection seems to develop in relation with the use of tension sutures, and in no circumstances should these be employed after excision of these ulcers.
- 5 The organisms are anaerobic streptococci and staphylococci, and it is assumed that the source of this unusual type of organism is probably from within the alimentary canal.

REFERENCE

- ¹ POAT, *Med Jour Australia*, 1930, 11, 398

A UNIQUE "SKINNING" ACCIDENT

ON Aug 30, 1934, Mrs N, aged 38, stepped off the cab of a lorry which her husband was driving, she fell down, and the back wheel of the lorry ran over the outer part of her left thigh and leg. The whole thickness of the skin with the subcutaneous tissue was stripped clean off the leg—from the groin to the ankle (*Fig 262*) On admission to the local hospital, a large flap of skin was found

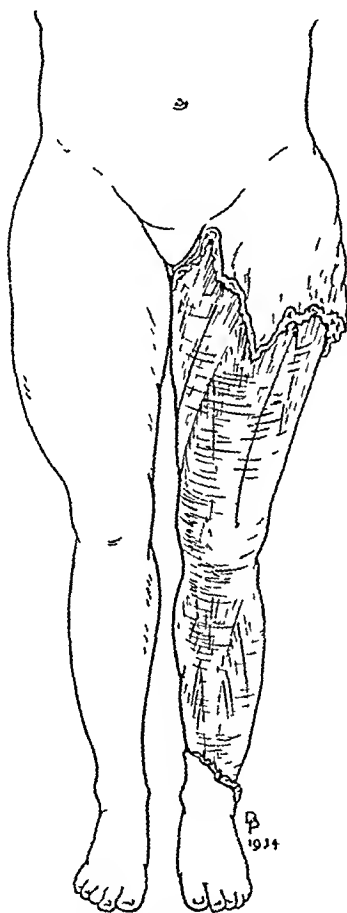


FIG 262 —Showing how the skin and subcutaneous tissue were stripped from the patient's leg

attached to the gluteal region. This was cut off. The patient suffered from a severe degree of shock, from which she rallied to some extent after continuous saline infusion. On Sept 4 her condition was so far improved that it was considered safe to amputate, but she died a few hours later.

NOTE—The skin must have been pinched along the outer side of the leg and split on the inner side. The whole thickness of the skin and subcutaneous tissue had been stripped off down to the deep fascia, through which the muscles could be plainly seen.

REVIEWS AND NOTICES OF BOOKS

The Origin of Cancer By J P LOCKHART-MUMMERY, M A, M B, B C (Cantab), F R C S, Senior Surgeon, St Mark's Hospital, etc Large post 8vo Pp 150 + x, with 29 illustrations 1934 London J & A Churchill 10s 6d net

THE transmutation of one or more of the cells of the body into a cell with unchecked and limitless powers of proliferation appears to be the event which determines the origin of cancer. The transmutation is either the effect of changes in the intracorporeal environment of the cell, or is a primary change within the cell itself. Mr Lockhart-Mummery's book states the case for the second of these two views. His theory, first put forward in 1932, is that if the growth-rate of somatic cells is controlled by certain genes contained in the nucleus of the cell, and "if these genes have been mutated in such a way that the normal rate of growth is increased, that is to say if mitosis of the cell occurs at more frequent intervals than is normal for that particular tissue, then it follows that a tumour must result" (page 37).

The author frankly admits that the 'gene' is a concept only, but in his view biology is reaching the position of physics, which has long dealt familiarly with such ultra-sensual concepts as the electron. He claims for the pathologist the freedom long conceded to the poet and lately seized by the physicist.

As imagination bodies forth
The forms of things unknown, the poet's pen
Turns them to shapes, and gives to airy nothing
A local habitation and a name

The question arises whether it is necessary, in order to account for an acceleration of the rate of multiplication of a cell or group of cells, to assume an essential change in the machinery of the cell, a gene mutation. Small external interferences or a mere fall of temperature may make a clock go faster, apart from any permanent alteration in its machinery. Is not the gene theory too long a jump beyond the known facts—hypothesis, in fact, rather than a theory?

Mr Mummery admits "Very marked variations may occur as the result of changes or abnormalities in the environment, as, for instance, when a child is born without a limb, or part of a limb, due to intra-uterine damage from adhesions, etc. Or when as the result of disease abnormalities of structure or function occur. Such abnormalities are clearly the result of the environment, but at present we cannot always distinguish between changes in the adult structure due to factors in the environment, and changes due to the genes which have been inherited" (page 74).

It would seem simpler, and therefore more scientific, to assume that in the cell about to be cancerous some permanent and generally irreversible change, not necessarily a gene-mutation, is produced by a change in the environment. In this connection Sampson Handley's demonstration that precancerous areas are areas in which lymph-stasis has been set up, usually by previous lymphangitis, must not be forgotten. It is true, as Mummery points out, that the lymph-stasis theory does not offer any precise explanation of the intracellular events which change a normal epithelial cell into a cancerous one, though Handley suggests that the cell in a condition of lymph-stasis is cut off from the chemical influence of the rest of the cell-community, and consequently reverts to its ancestral condition as an autonomous unicellular organism.

The strongest argument in favour of the gene-mutation theory of cancer is derived from a study of the tumours of homologous twins. The facts about these tumours are certainly mysterious and arresting. Taking Burkhard's case of the twins who both developed cancer of the left breast simultaneously, it would appear that in the original ovum which divided into a twin-ovum all the arrangements for an eventual and predestined cancer of the left breast were already perfected without regard to the post-uterine life history of the twins concerned.

Surely, however, these predestined cancers must form but a small fraction of the total of malignancy. The occupation cancers, the cancers due to infections such as syphilis or to trauma or exposure to light, are obviously excluded from them. They hardly supply a broad enough basis for rejecting the belief that as a rule the conditions leading to cancer are first established during the post-uterine life of the individual.

Enough has been said to show that Mr Lockhart-Mummery's book supplies much food for thought, especially for those who, like Sir Thomas Browne, love to pursue their reason to an *O altitudo*. It must be added that the author has expressed himself in clear and lucid terms which leave no doubt as to his meaning, and that even those who disagree with the book's conclusions must admit its intellectual appeal before recording a 'not proven' verdict.

Surgical Anatomy and Physiology By NORMAN C LAKE, M D, M S, D Sc (Lond), F R C S, Senior Surgeon, Charing Cross Hospital, etc., and C JENNINGS MARSHALL, M D, M S (Lond), F R C S, Surgeon, Charing Cross Hospital, etc. Demy 8vo. Pp 888 + v, with 238 illustrations. 1934. London. H K Lewis & Co Ltd. 30s net.

THE authors have considerably exceeded the scope of their work as set out in the preface, for besides surgical anatomy and physiology the book contains a good deal of pathology and much treatment, both operative and non-operative. There is an excess of operative detail, and even if it is admitted that a general outline of common operations should be included in a work on anatomy, it is open to question whether some of the operations chosen for description are in general use. Much of the physiology is biochemical, describing methods of investigation of use in diagnosis of disease. The inclusion of such material appears a helpful innovation, but care should be taken that only views which have stood the test of time and which are generally accepted should be included. Such a book is no place for theories which may be exploded long before the edition in which they appear is out of print.

Some of the figures are original and helpful, but others 'after' well-known text-books are inaccurate. We have compared Figs 49 and 117 with the originals in Cunningham's *Anatomy*, and it is difficult to realize that they are copies, and in each are obvious inaccuracies.

The authors set out upon a well-intentioned and much-needed project, and it is to be hoped that at an early date they will take advantage of a second edition to correct glaring errors which, in its present form, debar what might be a really useful book from being safely included in the library of a student, who will probably be unable to sift the wheat from the chaff.

La Pratique chirurgicale illustrée By VICTOR PAUCHET. Fasc XIX. Super royal 8vo. Pp 295, with 249 illustrations. 1934. Paris. G Doin et Cie. Fr 70.

THIS volume contains twenty-one articles, ten of which deal with resections of colon and rectum, closure of colostomy, colo-vesical fistula, vesico-vaginal fistula, and congenital malformations of the anus. The remaining subjects dealt with are retropharyngeal abscess, cholecystostomy, anastomotic ulcer, extra-mucosal pyloroplasty, gastrectomy, spina bifida, and medullary grafting for pseudarthrosis. Dr Pauchet and Dr P Le Gac each contribute eight articles, Dr G Luquet two, Drs A Tierny, G Pascalis, and J Seneque one each.

The high standard of excellence reached in previous volumes is maintained, thanks to the excellent illustrations by Monsieur S Dupret. Articles which call for special comment are those on the method of extra-mucosal pyloroplasty (G Luquet), closure of left-sided colostomy (P Le Gac), and the Hartmann operation for low pelvic colon growths (G Luquet). Dr Luquet employs a median incision for the Hartmann operation. We have found a long oblique iliac incision to give better access and facilitate the operation in a considerable number of cases, and we like to divide the upper end of the colon outside the abdomen at the end of the operation instead of at the commencement.

A simple perineal excision of the rectum on a magnified Whitehead principle is described for tuberculous ulceration of the rectum. The occasions when this is either desirable or possible must be few. If tuberculous ulceration is so extensive as to justify a radical excision it is usually associated with peri-rectal inflammation, fixation of the rectum, and often with inflammatory invasion of the ischio-rectal fossæ, etc., conditions most unfavourable for this type of operation.

X-ray and Radium Injuries Prevention and Treatment By HECTOR A. COLWELL, M.B., Ph.D., M.R.C.P., D.P.H., and SIDNEY RUSS, C.B.E., D.Sc., F.Inst.P., the Barnato Joel Laboratories, Middlesex Hospital. Demy 8vo. Pp. 212 + vii. Illustrated. 1934. London. Oxford University Press. 14s. net.

THIS is the only text-book of its kind in the English language. The outstanding features of these injuries are presented in a form which will be very helpful, not only to all radiologists but to all medical practitioners, and as references to the literature on the subject are freely given, the student who wishes to pursue the subject further will find this book invaluable.

After the opening chapter, which deals with protection, and in which full credit is given to the work of the X-ray and Radium Protection Committee of this country, the subject is treated in a series of chapters dealing with the various systems of the body. Naturally the skin takes precedence, and the story of the damage done to early X-ray workers and patients as a result of exposure to radiations is briefly retold. The effects of X-rays and radium on the skin are all carefully described. The various skin reactions, both early and late, are fully dealt with, and special mention is made of the work of Gillies and McIndoe in the treatment of chronic radio-dermatitis and radio-necrosis.

In succeeding chapters the effect of X and gamma radiations on the respiratory tract, the circulatory system, the alimentary tract, the generative system, the urinary tract, the nervous system, and bone are dealt with. Finally, chapters on radiation sickness and poisoning by radio-active substances complete a small volume in which a mass of accurate information is presented to the reader.

This country was the first to recognize the need for a system of adequate protection in connection with X-ray and radium work. The recommendations put forward by the X-ray and Radium Protection Committee have now received international recognition and are in general use.

The readers of this book will realize how much pioneers suffered, and how much has been done to make the work of the present-day radiologist safe from similar tragedies. It is a book which can be recommended to all radiologists and as a mine of information to those interested in radiological research.

Die Wirbelgelenke By Priv.-Doz. Dr. MAX LANGE (Munich). 10 × 6½ in. Pp. 121 + vi, with 59 illustrations. 1934. Stuttgart. Ferdinand Enke. RM. 7.

AN excellent and concise account of the vertebral articulations and of their appearance in X-rays which should be of real assistance in this difficult branch of radiology. The articulations have been examined by taking X-rays of specimens as well as by clinical investigations, and the angles necessary to show up the different parts of the vertebræ are illustrated by photographs which are in many cases clarified by a diagram. The pathological conditions are treated in a similar manner.

Narkose zu operativen Zwecken By Dr. HANS KILLIAN (Freiburg i. Br.). Super royal 8vo. Pp. 406 + viii, with 165 illustrations. 1934. Berlin. Julius Springer. Paper covers, RM. 24, bound, RM. 26.80.

DR. KILLIAN's book on anaesthetics, based on his own experience and supplemented by his wide knowledge of the international literature on the subject, covers the whole field of the theory and practice of anaesthesia. Exhaustive references to the bibliography given at the end of each section render the book a valuable source of reference. The theories of anaesthesia and the physiological changes that occur in the various organs are discussed with more detail than is generally found in works of this nature, the chapter dealing with the technical side of the subject, however, follows more conventional lines, whilst the descriptions of the actual methods of administration—contrasting strangely in this respect with the wealth of detail devoted to the other sections of the book—are patently inadequate. In the chapter dealing with the choice of anaesthetic there is much with which English anaesthetists will disagree, though, as Dr. Killian explains, the choice in Germany is limited by the lack of skilled anaesthetists: thus little use is made of nitrous oxide or nitrous oxide and oxygen, and it is probably for this same reason that the author condemns the use of chloroform even in obstetrics. Evipan, amongst other recently introduced drugs, is described and is

recommended as a safe anæsthetic for many operations. The volume concludes with a description of many of the anæsthetic machines in common use, and it is to be regretted that, whilst this book is in other respects completely up-to-date, obsolete models of English apparatus should be illustrated and described.

Röntgendiagnostik der Knochen- und Gelenkkrankheiten By Professor Dr ROBERT KIENBOCK (Vienna). Large 8vo. Part 1. Differentialdiagnose der geschwultstigen Knochenkrankheiten. Pp 104, with 26 illustrations. Part 2. Knochenechinokokkose. Pp 105-192, with 26 illustrations. 1933. Berlin and Vienna. Urban & Schwarzenberg. Part 1, RM 8 50, Part 2, RM 7 60.

THE first two parts of a series to be devoted to the X-ray diagnosis of bone and joint disease include (1) New growths, and (2) Echinococcus cysts of bone. The former is difficult because the classification and terminology differ so much from that used in this country. The latter is of little interest because of the rarity of the condition in this country. The illustrations are good, but are less numerous in proportion to the text than would be expected in a book on X-ray diagnosis.

Acute Intestinal Obstruction By MONROE A. McIVER, M.D., Surgeon-in-Chief, Mary Imogene Bassett Hospital, Cooperstown, N.Y. 10¹ x 7 in. Pp 430 + xviii, with 62 illustrations. 1934. New York. Paul B. Hoeber Inc. \$7 50.

A MONOGRAPH on acute intestinal obstruction is no longer a tedious presentation of the various types and mechanical forms of the condition. The problems presented by intestinal obstruction have been investigated from a variety of angles during the last decade and there has been a definite need for a work which attempted to evaluate the results obtained and correlate them with the clinical and pathological material. It is therefore gratifying to find a monograph on the subject published by an experienced surgeon who has previously made experimental researches which have contributed to a clearer understanding of the fundamentals of acute obstruction.

The book is divided into three sections. Part I gives a general picture of the condition, Part II deals with diagnosis and methods of treatment, and Part III discusses the experimental work which has been carried out to determine the cause of death from intestinal obstruction. The work is well balanced and the material is admirably presented. The author has evidently been careful not to allow enthusiasm to lengthen unduly any section of the book, and the chief criticism which might be applied is that he has not sufficiently stressed the practical importance of the changes taking place in the circulation and body fluids in acute obstruction—a subject on which he and his associates have carried out valuable researches. In consequence one feels that the overwhelming importance of the pre-operative treatment is not given the attention it warrants. The indications for ‘decompression’ of the stomach and intestines by a duodenal tube are examined critically and their values reasonably assessed.

The question of anæsthesia is not given the importance it deserves. The point is made that patients with a late obstruction are in a condition of shock which resembles that seen after severe traumatic injuries, but there is no suggestion that gum saline or blood transfusion might be beneficial. To suggest that local anæsthesia, with all the limitations it involves, is often the anæsthetic of choice is to admit that the pre-operative measures may have such a limited success in restoring the blood volume that the use of spinal anæsthesia, undoubtedly the ideal anæsthetic, is inadvisable. The remarks on shock following anæsthesia which are included in the chapter on complications would have made a useful introduction to the section in which anæsthesia is considered. Otherwise the chapters on treatment are admirable and well worth reading by every practising surgeon.

The chapters on “The Cause of Death” are very well done. An excellent and studied summary of the methods and results of experimental researches is given. The theory that the cause of death in many cases is a toxæmia is carefully examined and the relevant experimental work quoted, but one is glad to see that room is found for the view of those workers who do not believe that a toxæmia is the lethal factor in low obstructions. There is much to be said for the view that “the stagnation of blood and fluid in the splanchnic area may lead to peripheral circulatory failure and shock in this type of obstruction.”

Apart from the minor points mentioned the work is excellent, and the author and publishers are to be congratulated on so admirably filling a gap in surgical literature.

Diathermy in General Practice By ERIC PAYTEN DARK, M.C., M.B., Ch.M. (Syd.), late Radiographer, Royal Prince Alfred Hospital, Sydney With special chapters by F. A. MAGUIRE, C.M.G., D.S.O., M.D., Ch.M. (Syd.), F.R.C.S., F.R.A.C.S., and GUY P. O. PRIOR, L.R.C.P., M.R.C.S. Second edition Demy 8vo Pp 219 + xvi Illustrated 1935 Sydney Angus & Robertson Ltd 17s 6d net

THIS book is now appearing in its second edition. As it is intended to be of assistance to the general practitioner, who, presumably, has little knowledge of electro-technics, the opening chapters on the action of diathermy machines are not sufficiently instructive. This part of the work could be very much improved or should be omitted altogether. Simple instruction in the control of the diathermy machine would suffice. A large part of the rest of the book is made up of case records. These are of little interest to the general practitioner. A simple classified table showing the percentage of cures and the number of applications of treatment would satisfy and economize space. There are several excellent photographs showing the method of applying electrodes to various parts of the body. A unique feature is a chapter on failures. It would appear that the author had only 8 failures out of 91 cases. This ratio is much better than is obtained by most electrotherapists in this country. No mention is made of the cutting current for surgical work nor of short-wave or ultra-short-wave diathermy. No doubt the author considers that these should be left to the expert. One feels that the treatment of gonorrhœal, gynecological, and mental conditions should be left to the expert also. This would appear to be the opinion of the writer of the foreword also, who states, "the technicalities of correct diathermy and the time required for each case make it probably best that its use should mainly lie in the hands of those specially competent."

Benign Tumours in the Third Ventricle of the Brain. Diagnosis and Treatment By WALTER E. DANDY, Adjunct Professor of Surgery, the Johns Hopkins University. 10 x 6½ in. Pp 171 + viii, with 120 illustrations. 1934 London Baillière, Tindall & Cox 22s 6d

THIRD-VENTRICLE tumours have long constituted one of the most intriguing group of intracranial neoplasms, partly on account of their interesting and often puzzling clinical features and partly because of their unusual histopathology. They have not, however, been regarded commonly as favourable tumours for surgical intervention, despite their benign nature. Dandy has demonstrated, however, that the third-ventricle growths are amenable to surgery and has collected an impressive total of 21 cases in which the tumour was removed, with a mortality rate of 33.3 per cent. In this present monograph Dandy has given us an intimate presentation of the clinical histories of these cases, together with notes of 47 cases taken from the literature, all of these latter were post-mortem findings, none having been correctly diagnosed during life. Dandy has analysed in detail the neurological features and has emphasized the great difficulty in arriving at a correct localization from the symptoms and signs alone. Several clinical features are indicated as being suggestive: (1) Loss of pupillary reflexes, (2) Intermittent attacks of various kinds, namely, (a) headache, (b) dizziness on alteration of posture, (c) bilateral weakness or numbness, and (d) loss of vision, (3) Possibly, loss of hearing for high tones, (4) Bilateral ptosis and ophthalmoplegia. It is obvious that those signs not of hydrocephalic origin are merely indicative of pressure upon the quadrigeminal plate. Headache, vomiting, and papilloedema are usually intense and early, and the intermittency of the first symptom often points to a ball-valve obstruction to the ventricular outflow. It is clear from the author's evidence that ventriculography offers a diagnostic short-cut which is safe and unmistakable. There always appears an enlargement of both lateral ventricles, air may or may not pass from one lateral ventricle to the other. No air as a rule enters the third ventricle, when, however, the obstruction at the foramina of Monro is incomplete or intermittent, the third ventricle may appear in the skiagram: (1) Not dilated, because it is behind the obstruction, (2) Showing a filling defect which outlines the posterior and perhaps the inferior part of the tumour, and (3) Small, upright, and mesially placed in the antero-posterior view.

From the pathological standpoint Dandy recognizes two groups of third-ventricle tumours: (1) Colloid cysts, which grow from the antero-superior aspect of the ventricle, the lining epithelial cells are frequently ciliated, and Dandy supports Sjovald in believing that they are of vestigial origin. (2) A heterogeneous group of tumours including ependymal gliomata,

choroid-plexus tumours, epidermoids (tumeurs perlees), and several other indeterminate histological types

Dandy has employed two chief surgical approaches. A posterior 'pineal' route and a frontal or 'hypophysial' approach are the two usual operative attacks. In the case of cysts of the septum pellucidum, a third or mid-sagittal approach through the corpus callosum has also been employed. The frontal attack—used mainly when the bulk of the tumour occupies the forepart of the ventricle—necessitates the resection of an oval or transverse segment of the frontal lobe. Dandy says that no loss of function, mental or physical, has resulted from these frontal-lobe extirpations, and, so far, post-operative epilepsy has not been encountered.

This contribution makes an important landmark in the progress of neurosurgery. It is issued with a wealth of illustration and lavish disregard of expense characteristic of the American monograph-writer.

St Bartholomew's Hospital Reports Edited by LORD HORDER, R. G. CANTI, W. SHAW, C. F. HARRIS, H. H. WOOLARD, R. C. ELMSLIE, W. G. BALL, G. EVANS, and J. P. ROSS. Vol. LXVI. Demy 8vo. Pp. 363 + xlv. Illustrated. 1933. London. John Murray. 21s. net.

THE staff of St Bartholomew's Hospital, both clinical and scientific, have done some valuable team work on the autonomic nervous system in its relation to medicine and surgery. This volume contains a valuable symposium on the sympathetic nervous system which will interest physicians as well as surgeons, because it includes a case of paravertebral injection for angina pectoris, but perhaps the most interesting paper in this collection is that by Mr R. E. Norrish on the anatomy of the sympathetic. As has been previously demonstrated by other writers on the question, this paper shows that the great variation in the minute anatomy of the sympathetic very much hampers scientific deductions from experimental and operative procedures.

Dr Branson's review on the health of the nursing staff for a period of ten years is a particularly valuable contribution, partly because, as its conclusions are drawn from a large nursing staff, fairly exact deductions may be formed from them. Scientific observations of this kind have been long over-due, and it is a feather in the cap of the oldest hospital in London that it should be among the first to include in its reports papers of this kind. The next paper on "Pre-Reformation Nurses in England," by Miss Margaret Hart, still further demonstrates the tendency of this publication to be in the van. This is, I believe, the first paper written by a woman which has been published in the *Reports*, and is of importance because it was awarded a prize by the National League of Nursing Education of America.

Mr G. M. Lloyd writes charmingly of the life and work of Percivall Pott, whose heroism when he fell from his horse in the Old Kent Road and sustained a compound fracture of the tibia must have been even more remarkable in that day than it would be now, owing to the dangers inherent in such an injury. The reviewer would have preferred at the head of the article the delightful portrait of Pott by Romney in the Council Room of the Royal College of Surgeons, in place of that by Reynolds in the Great Hall at St Bartholomew's, which shows him at a more mature and less attractive age.

BOOK NOTICES

Surgery of a General Practice By ARTHUR E. HERTZLER, M.D., Chief Surgeon, Halstead Hospital, etc., and VICTOR E. CHESKY, M.D., Chief Resident Surgeon, Halstead Hospital. Large 8vo. Pp. 602, with 427 illustrations. 1934. London. Henry Kimpton. 42s. net.

Fuss und Bein ihre Erkrankungen und deren Behandlung By Prof. Dr. med. GEORG HOHMANN (Frankfurt a. M.). Second edition. Royal 8vo. Pp. 380 + x, with 326 illustrations. 1934. Munich. J. F. Bergmann. Paper covers, RM. 24, bound, RM. 25.80.

- Modern Advances in Diseases of the Throat** By ARTHUR MILLER, F R C S (Ed), D L O, Surgeon for Diseases of the Ear, Nose and Throat, French Hospital, London, etc Medium 8vo Pp 120 + xii, with 40 illustrations and 1 coloured plate 1934 London H K Lewis & Co Ltd 10s 6d net
- Cirugia gástrica** By Dr MANUEL CORACHAN (Barcelona) Vol I Crown 4to Pp 775 + xiv, with 374 illustrations 1934 Barcelona Salvat Editores, S A No price given
- Bassini's Operation for the Radical Treatment of Inguinal Hernia** By Prof ATTILIO CATTERINA, University of Genoa Obl Imperial 4to Pp 58, with 16 coloured plates by Orazio Gaigher, M D 1934 London H K Lewis & Co Ltd 30s net
- Röntgendiagnostik der Knochen- und Gelenkkrankheiten** By Prof Dr ROBERT KIENBOCK (Vienna) Part 3 Gelenksosteomatose und Chondromatose Large 8vo Pp 228, with 194 illustrations 1934 Berlin and Vienna Urban & Schwarzenberg RM 22 50
- Operationstaktik bei Erkrankungen der Gallenwege** By W M STERN (Paris) and R FOURCHE (Nancy) Translated by Dr E HAYWARD (Berlin) Super royal 8vo Pp 256 + viii, with 203 illustrations 1934 Berlin and Vienna Urban & Schwarzenberg Paper covers, RM 12, bound, RM 13 50
- The Science and Practice of Surgery** By W H C ROMANIS, M A, M B, M Ch (Cantab), F R C S (Eng), F R S (Edin), Surgeon and Lecturer on Surgery, St Thomas's Hospital, etc, and PHILIP H MITCHNER, M D, M S (Lond), F R C S (Eng), Hon Surgeon to H M the King, etc Fifth edition Royal 8vo In two volumes Vol I, General Surgery Pp 789 + 75 pp of index Vol II Regional Surgery Pp 962 + 75 pp of index 758 illustrations 1934 London J & A Churchill 14s each volume
- Visceriti e Perivisceriti digestive addominali croniche** By FRANCESCO ZAGARESE Crown 4to Pp 359, with 60 illustrations 1934 Bologna Nicola Zanichelli L 40
- Radium and Cancer** By H S SOUTTAR, D M, M Ch (Oxon), F R C S, Surgeon, London Hospital 8½ in × 6½ in Pp 387 + xiii Illustrated 1934 London William Heinemann (Medical Books) Ltd 21s net
- Localization of Function in the Cerebral Cortex** By Various Authors Vol XIII of a Series of Research Publications Editorial Board S T ORTON, M D, J F FULTON, M D, T K, DAVIS, M D Medium 8vo Pp 667 + xvi, with 171 illustrations 1934 Baltimore The Williams & Wilkins Company (London Bailière, Tindall & Cox) 36s net
- Westminster Hospital Reports** Edited by STANFORD CADE and J V PULVERTAFT Vol XXII (1929-33) Demy 8vo Pp 428 + iv, with 8 illustrations 1934 London H K Lewis & Co Ltd 7s 6d net
- The Newcastle upon Tyne School of Medicine, 1834-1934** By G GREY TURNER, assisted by W D ARNISON Large 8vo Pp 224 + xii Illustrated 1934 Newcastle upon Tyne Andrew Reid & Company Ltd 10s net

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IPSISSIMA VERBA

BY SIR D'ARCY POWER, K B E, LONDON

IV. TWO PRE-HUNTERIAN OPERATIONS FOR ANEURYSM

(Concluded)

THE second operation recorded by Richard Wiseman is in the 1676 edition of his *Severall Chyrurgical Treatises* and is Observation 5, page 76, of "Of An Aneurisma" —

"A man being much afflicted with a Tumour in his right Arm, occasioned by the pricking of an artery in letting blood having tried the endeavours of several of our Profession unsuccessfully, some whereof had proposed the taking off this Arm, at the last I was sent for, and met Mr Arris and Mr Hollier there Mr Gardner was his Chirurgeon The Tumour was large much inflamed and painfull, with little or no pulsation, but the Accident which gave rise to the Tumour enough confirmed it an Aneurisma My opinion was, that a Tumour so inflamed was not capable of such Bandage whereby they might hope to return the blood back into the Artery Nor indeed was it reasonable to suppose that such a putrefactive heat as seemed to be enclosed in that Swelling was capable of being thrust back or retained long there, without making its own way by a Gangrene, and of what danger such an Eruption might be to the Patient, if a Chirurgeon were not at hand, I left to their consideration Then in order to the removal of the Tumour there were but two ways, viz Amputation of the Arm or cutting into the Tumour, and making Deligation of the Artery, which latter I commended to them They assented to the Deligation, onely the Patient desired a day or two to prepare himself We applied Empl e bolo with a moderate Bandage to restrain the increase of the Tumour the while

"The fourth day after we met again, and, having all things ready, the Patient was placed in a Chair towards the light I took off the Dressings, and made a Ligature four fingers breadth above the Tumour, on which Mr Hollier made a gripe Some other held the Hand and lower part of the Arm, whilst I made an Incision down the length of the Tumour That done, I threw out the grumous blood with my fingers and cleansed the wound with a sponge Then desiring Mr Hollier to slacken his hand, upon which the artery discovered itself by the

blood spurting out, I passed my Needle under the upper part of the Artery and tied that, and cut off the end of the Ligature Near the Wound made in the Artery by letting blood there was a cartilaginous body formed, which hindered my coming to the Artery, I cut it away, then passed my Needle, and made a second Ligature We dressed it up with Pledgits spread with the common Digestive extractin dipped in pulv Galeni, and applied Empl diachalcit malaxed with oil of roses and rowled it up to the ease of the Patient, then put him into his bed, and prescribed him an Anodyne draught to take that night The next day I visited him, and found him in much ease he had slept well with half the draught [which was] designed for him, and was not troubled with those fainting fits which he had been subject to before the Operation

"The third day we took him out of his bed to dress his Arm In taking off the Dressing we found all safe, and the Lips of the wound tending to digestion Mr Hollier, who held the arm above, seeing no blood flow from the wound, for experience sake griped it harder, upon which the blood of a sudden dropped from the fleshy parts and Capillaries in great drops, as if it had been pressed out of a Sponge I wondered at the manner of its bleeding, not minding what he had done, but he slackning his hand, it ceased as soon Which may show the ill consequence of over-hard binding in the dressing of Wounds, Ulcers &c We dressed the Wound with the Digestive as before, and so continued it till the Wound was well digested From that time we dressed it with Sarcoticks, rubbed the loose flesh with the Vitriol-stone, and hastened the cicatrizing of it with aq medicamentos [lime water] &c I saw the Ligatures fall off and the Wound cicatrizing, then left it to his Chirurgeon, it being at that time indeed almost cicatrized The patient made me a visit a while after and showed me that he could use that Arm as well as the other"

Mr Wiseman's two assistants at the operation were noteworthy surgeons Mr Edward Arris, himself the son of a barber surgeon, was born in 1591 and died in 1676 He was buried in St Sepulchre's Church, where his memorial is still to be seen He was a man of wealth and position, living in St Bartholomew's Hospital, a Governor, but never Surgeon, and an Alderman of Bridge Ward without He was prolific, for his wife had twenty-three children Liberal-minded and generous, his good qualities were not shared by his eldest son Robert, who, at any rate in his later years, proved himself to be mean and cantankerous Excuses perhaps may be made for him because he became blind and was in such reduced circumstances that he was given a pension

The name of Mr Alderman Arris is perpetuated in the Arris and Gale Lectures which are still given annually at the Royal College of Surgeons of England on subjects relating to human anatomy and physiology, the lecturer being called 'Professor' and the honorarium being roughly £10 a lecture The record of the foundation of the lectureship runs as follows, and it may be noted that the bequest was at first anonymous —

"27th October 1645,—This day Mr Edward Arris acquainting this Court (of the Company of Barbers and Surgeons) that a person, a friend of his (who desired his name to be as yet concealed) through his greater desire of the increase of knowledge of Chirurgerie did by him freely offer to give unto this corporation for ever the sum of 250 *li* to the end and upon condition that a human body be once in every year hereafter publicly dissected and six lectures thereupon read

and if no human body may be had nor conveniently dissected in one year then the Company to distribute one half of the sum of the usual charges of a public anatomy to our own poore and the other half to the poore of St Sepulchre's The said worthy overture is thankfully accepted by this Court and that for the performance thereof a Rent charge of xx *h* per annum be granted out of our lands at Holborn Bridge "

A year later "This Court doth order that all the approved Chirurgeons according to law shall appear at all publique anatomyes for the time to come in a flatt capp, upon the penalty of 3s 4*d* and all the rest of the Livery in a Hatt " The surgeon's 'flatt capp' was green with a white ribbon round it

Two other benefactions were made by Mr Alderman Arris He bought back and gave to the Company the Henry VIII's cup which is now the chief treasure of the Barbers' Company It had been sold during the Commonwealth, and he made a skeleton of Canonbury Bess, a greatly wicked woman, who was properly hanged Her skeleton was used for many years by the Lecturer on Anatomy—Clopton Havers amongst others—and it appears in the last plate of Hogarth's "Idle Apprentice "

Thomas Hollier, Wiseman's other assistant, was successively Surgeon for Scald Heads (1638), Surgeon (1644), and Cutter for the Stone (1663) at St Thomas's Hospital Mr F G Parsons states that he received £36 a year as Surgeon and an additional £15 as Lithotomist He resigned his hospital appointments in 1670 and was succeeded by his son His house was blown up on May 26, 1676, to prevent the great Southwark fire spreading to the hospital buildings He operated upon Mr Samuel Pepys, the diarist, on March 26, 1658, and was Warden of the Barber Surgeons' Company when Wiseman was the Master in 1665

It may be noted that Wiseman opened the sac of the aneurysm, turned out the clot, and ligatured the artery immediately above the sac, Hunter tied the artery at some distance from the sac, which he left untouched The method of cure therefore, was radically different in the two methods

THE ETIOLOGY OF THE VASCULAR SYMPTOMS OF CERVICAL RIB *

By D M BLAIR

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IN a small proportion of clinical cases of cervical rib the symptoms are predominantly of a vascular nature, motor and sensory symptoms being absent or very slight. Recently Telford and Stopford¹ suggested an anatomical basis to account for this predominance of vascular symptoms. In a number of dissecting-room subjects without cervical rib they examined histologically the lower trunk of the brachial plexus in the region where it lay on the upper surface of the first thoracic rib. In the eighth cadaver investigated they found in the inferior part of the lower trunk of the plexus a distinct and separate bundle of unmyelinated fibres, which they interpreted as the sympathetic fibres passing to the upper limb by way of the lower trunk and which had not yet become incorporated with and intermingled amongst the other fibres of the lower trunk, in other words, the fusion of the 'sympathetic' fibres with the lower trunk was in this instance at a more distal point than usual. In the other specimens, at the site where the lower trunk of the plexus lay on the first rib, the unmyelinated fibres were found already scattered amongst the other fibres of the trunk, although these authors noted a slight tendency for the unmyelinated fibres to reside principally in the peripheral parts of the trunk. Telford and Stopford observe that, in the special instance described, the separate bundle of unmyelinated fibres in the inferior part of the lower trunk of the plexus would evidently be more immediately exposed than the motor and sensory fibres to the risk of pressure by the subjacent rib. They further postulate that, given the same condition in the lower trunk of the plexus, the symptoms produced by a cervical rib would be predominantly vascular and would be of the nature of chronic arterial spasm induced by prolonged *irritation* of this separate exposed bundle of 'sympathetic' fibres by the pressure of the cervical rib. This prolonged spasm of the arterial stems would cause constriction or even obliteration of their vasa vasorum, with consequent nutritional changes in the arterial walls which might ultimately lead to thrombosis. That the vascular effects in these cases of cervical rib are usually confined to the arteries distal to the axillary artery is correlated by these authors with the fact that, whereas the subclavian and axillary arteries are supplied with sympathetic nerves directly from the sympathetic chain,

* Communicated to the Anatomical Society of Great Britain and Ireland, Summer Meeting, June 22, 1934

the more distal arteries in the limb receive their sympathetic innervation from the adjacent main nerves of the limb. Of this distal nerve-supply, those nerve-fibres which issued via the lower trunk of the brachial plexus would be affected, as they lie in the form of a separate bundle in the inferior part of the lower trunk immediately related to the rib.

We had recently the exceedingly rare opportunity of examining histologically the brachial plexus from a case of cervical rib in which the vascular effects were pronounced. For the specimen and the clinical history we are indebted to Mr Ivor Back.

CASE REPORT

(W McKISOCK)

The patient, aged 37 years, a married woman without children, seven months prior to admission to hospital experienced painful tingling of the ring finger of the right hand. The nail of this digit was removed without subsequent improvement in the symptoms. During the next few weeks pain progressively spread to involve the other digits of the right hand, the hand, and finally the forearm as well, never extending above the elbow. In this period pain was felt only when use was made of the right hand or forearm, and disappeared when the limb was rested, but by the time of admission to hospital the pain had become continuous. When the hands were placed in cold water the patient observed that the right hand became white and cold, the left remaining normal. On elevation of the hands above the head, the right hand became white, but if kept hanging by the side it grew bluer in colour and felt unusually hot; the left hand did not share in any of these phenomena. For twelve months before admission the patient had a severe cough, without expectoration, and had been losing some weight.

ON ADMISSION—The patient complained of continuous severe pain, generalized and deep-seated, in the right hand and forearm. No cutaneous anæsthesia or paræsthesia or muscular wasting could be detected, particular attention being paid to the ulnar-nerve musculature. The right arm and forearm were each $\frac{1}{4}$ in less in circumference than those of the left side, the patient was normally right-handed, but had come to use the left more than the right owing to the pain. With the arms hanging by the side the right hand rapidly became blue and warm, whilst on elevation of the arms it at once lost its blueness and in a few seconds was white and cold. The left hand remained normal under these conditions. No accurate temperature records were taken. The right hand and forearm sweated on warming. No pulsation could be felt in the radial, ulnar, or brachial artery of the right side; pulsation in these arteries was normal on the left side. Pulsation was normal in the right axillary artery. Excessive pulsation was palpable in the right subclavian artery, so much so that (as is usual in these cases) subclavian aneurysm was suspected. On either side a cervical rib was palpable, more prominently on the right side. Radiological investigation revealed bilateral bony cervical ribs and extensive infiltration of both lungs characteristic of tuberculosis.

OPERATION—In view of the severity of the pain in the limb, it was decided to operate. At operation the subclavian artery was found to be quite free from pressure by the cervical rib, even when the arm was pulled down by the side. Pulsation in it was forcible and extended beyond the cervical rib and beyond the

scalene muscles. The lower trunk of the brachial plexus lay in immediate contact with the upper aspect of the cervical rib and the first dorsal contribution to the lower trunk was stretched taut as it passed upwards and laterally to the upper surface of the cervical rib. With the exception of its extreme anterior and posterior parts, the cervical rib (which extended from the seventh cervical vertebra to the first costochondral junction) was removed with its periosteum. The lower parts of the brachial plexus (lower trunk and first dorsal nerve) were then found to be quite free from pressure by the first thoracic rib.

At the end of the operation, faint pulsation could be detected in the radial artery. The next morning weak pulsation was palpable in the radial, ulnar, and brachial arteries. This pulsation increased slightly in intensity from day to day, but had not become normal (compared with the other side) by the eighth day, on which the patient died, the chest condition having lit up. The pain in the right upper limb, though lessened, had not been completely abolished by the operation. Complete post-mortem examination was not permitted.

HISTOLOGICAL EXAMINATION OF THE BRACHIAL PLEXUS

(D. M. BLAIR and F. DAVIES)

None of the arteries was available for examination. The specimen illustrated diagrammatically in *Fig. 263* was removed through the site of the operation incision. It comprised the trunks, bound together in prevertebral fascia, and the distal parts of the roots of the brachial plexus. The portion of the first dorsal root included

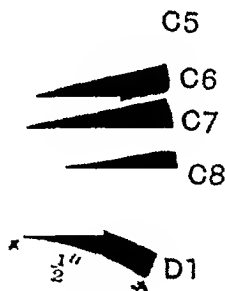


FIG. 263—Diagram of portion of brachial plexus removed. X-----X = 13.5 mm

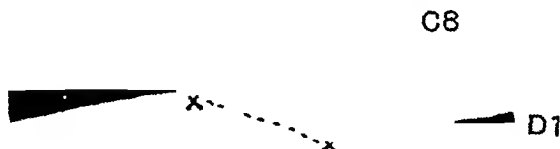


FIG. 264—Variation in length of first dorsal nerve contribution to plexus in dissecting room subjects. X-----X = 18 to 25 mm

in the specimen measured 13.5 mm. In a number of dissecting-room subjects the length of the first dorsal nerve contribution to the lower trunk of the brachial plexus, from the place where it left the first intercostal nerve to the place where it joined the eighth cervical nerve, was found to range from 18 to 25 mm (*Fig. 264*).

So at least the distal half of this portion of the first dorsal nerve was included in the specimen removed. This is of importance in relation to the fact that at no point along the first dorsal nerve or the lower trunk of the brachial plexus were we able to find an entering grey ramus.

The specimen was treated with a pyridene silver technique* which two of us (D M B and F D) have evolved in dealing with another problem, cut serially in transverse section, and examined microscopically from end to end. In only two of the roots of the brachial plexus were grey rami seen entering—namely, one in each of the seventh and eighth cervical nerves. The other rami joining the roots of the plexus must have entered the roots proximal to the specimen. *Fig 265* shows the entry of a grey ramus into the seventh cervical nerve. The ramus consists almost exclusively of unmyelinated nerve-fibres which are closely packed together with very little endoneurium and very few endoneurial nuclei between the fibres. Immediately on entry the unmyelinated nerve-fibres distributed themselves amongst the other nerve-fibres in the seventh cervical nerve. The same characteristics were observed in the case of the grey ramus entering the eighth cervical nerve. Numerous unmyelinated fibres were found scattered among the myelinated fibres even in the most proximal sections of all the roots of the plexus.



FIG 265—Entry of grey ramus into the seventh cervical nerve (60)

Fig 266 gives a general view of the homogeneous structure of the proximal part of the first dorsal nerve, while *Fig 267* is a higher-power view of a small area of the same section, near the centre, but not specially chosen. It is typically representative of the structure of the proximal part of the first dorsal nerve and shows numerous unmyelinated fibres scattered amongst the myelinated fibres, with very little endoneurium and very few endoneurial nuclei between the nerve-fibres. This is in contrast with the structure of the distal part of the first dorsal nerve, where it had come into close relation with the cervical rib. Here, as is illustrated in *Fig 268*, the endoneurium is increased in thickness and the endoneurial nuclei are much more numerous. The nerve-fibres do not form such a compact and 'clean' picture as in the proximal part of the nerve. The lower trunk of the brachial plexus shows the same histological features, particularly in its inferior portion (*Fig 269*), the thickened endoneurium and numerous endoneurial nuclei contrasting with the appearance of sections of the upper and middle trunks, in which the endoneurium is slight in amount and the endoneurial nuclei are few in number. The other roots of the plexus have a structure like that of the proximal part of the first dorsal nerve (with the exception of the number of unmyelinated nerve-fibres, as will be referred to below).

* This technique will be described in a forthcoming number of the *Journal of Anatomy*



FIG 266—Transverse section of proximal part of first dorsal nerve ($\times 37$)

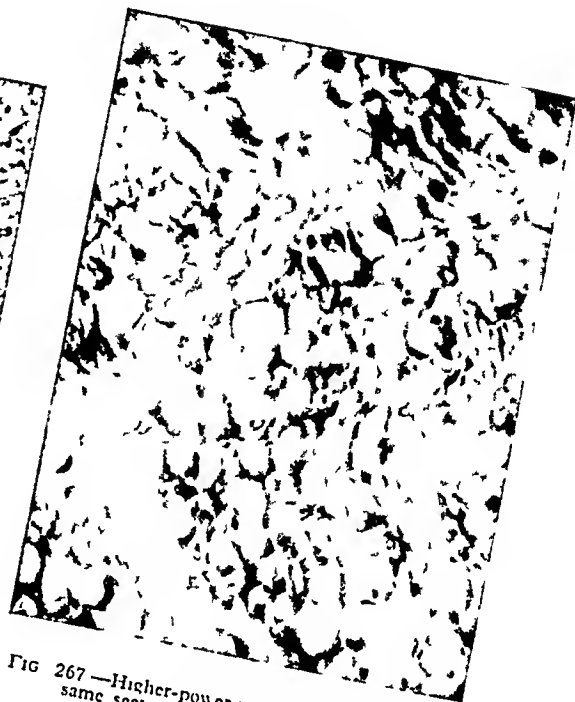


FIG 267—Higher-power view of central part of same section as *Fig.* 266 ($\times 375$)

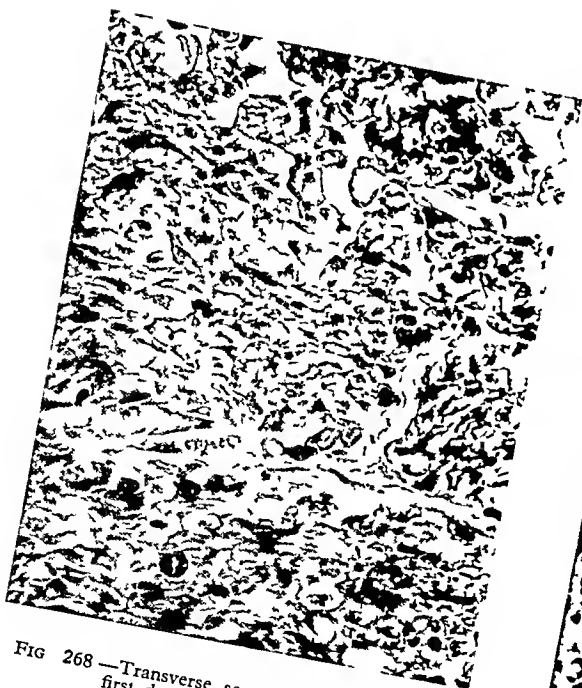


FIG 268—Transverse section of distal part of first dorsal nerve ($\times 375$)

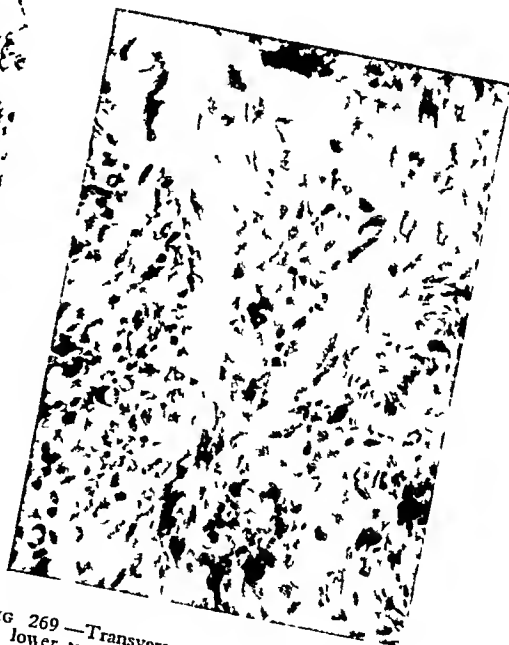


FIG 269—Transverse section of inferior part of lower trunk of brachial plexus ($\times 375$)

Fig 270 shows a low-power view of a section through the lower trunk of the brachial plexus. In the inferior part of the trunk is seen a distinct and separate collection of nerve-fibres which, as higher magnification (Fig 271) shows more clearly, consists of a number of small bundles. This collection of nerve-fibres corresponds in position with the separate bundles illustrated by Telford and Stopford in their drawing (Fig 413) in that it lies below the other fasciculi of the lower trunk and therefore in more immediate relation to the subjacent rib.

Examination of these small bundles of nerve-fibres with a high-power objective reveals that they consist mainly of unmyelinated fibres, as shown in Fig 272, which is a photomicrograph of the lower right bundle of Fig 271. Although not seen in the actual small area photographed, numerous large medullated nerve-fibres are also found in these bundles. As the photograph (Fig 272) shows, the unmyelinated fibres are loosely scattered in a fashion quite unlike the compact arrangement of the unmyelinated nerve-fibres in the grey rami described above. There is a large amount



FIG 270 —Transverse section of lower trunk of brachial plexus. Arrow points to separate collection of nerve fibres in inferior part of trunk ($\times 12$)



FIG 271 —Separate collection of nerve-fibres in inferior part of lower trunk ($\times 60$)



FIG 272 —High-power view of portion of separate collection of nerve-fibres in inferior part of lower trunk ($\times 375$)

of endoneurium and numerous endoneurial nuclei between the unmyelinated fibres. The photomicrograph, which was taken with a panchromatic plate and the deep-red (F) Wratten filter, shows many of the nuclei as black areas owing to deep

brown-black impregnation with silver. By the use of an infra-red plate and appropriate filters^{2,3} the nuclei are rendered clearly visible and do not mask the black unmyelinated fibres (Fig 273). On tracing this separate collection of nerve-fibres through the serial sections it was found to have an independent existence



FIG 273 —Same as Fig 272 Infra red plate
(375)

for only $\frac{1}{4}$ in. Both proximally and distally its nerve-fibres passed into the neighbouring fasciculi of the lower trunk and at once mingled amongst their nerve-fibres. This separate collection commenced $\frac{1}{2}$ in distal to the junction of the eighth cervical and first dorsal nerves and, as stated above, ran distally for only $\frac{1}{4}$ in. Therefore it cannot be regarded as a grey ramus joining the lower part of the brachial plexus at a more distal point than usual. It is merely a collection of nerve-fibres (mainly unmyelinated) effecting a re-grouping within the lower trunk.

Although no absolute counts were made, it was quite evident that the number of unmyelinated nerve-fibres in this separate collection formed only an infinitesimally small fraction of the total unmyelinated fibres in the lower trunk (see Fig 269). In this connection it might be noted that we observed the

lower trunk of the plexus to be much richer in unmyelinated fibres than the middle trunk, while the upper trunk of the plexus contained distinctly the fewest. Further, we were able to confirm the observation of Woollard¹ that the caudal roots of the plexus are richest in unmyelinated nerve-fibres. We noted no tendency for the unmyelinated fibres to reside principally in the peripheral parts of the lower trunk as Telford and Stopford described.

DISCUSSION

We agree with the view of Telford and Stopford that the clinical picture in these cases is one of *imitation* of the sympathetic (vasoconstrictor) fibres passing to the distal arteries of the upper limb, and not one of paralysis of sympathetic fibres, as other observers thought.

In the cases reported by Telford and Stopford thrombosis had taken place in the arteries of the limb distal to the axillary, and owing to the time necessary for canalization of the thrombus, several months elapsed subsequent to the removal of the cervical rib before pulsation returned in any of the large arterial stems. In the present case the rapid return of pulsation in the radial, ulnar, and brachial arteries after operation signifies that thrombosis had not supervened upon the arterial spasm. The increased tension of the arterial walls and the diminution of their lumen had alone been sufficient to render pulsation undetectable, at least by

the ordinary method of palpation. That pulsation in these arteries, however, did not immediately return to normal on removal of the cervical rib, and the incomplete disappearance of pain after this operation, suggest that the irritation of the sympathetic fibres is not due merely to mechanical pressure of the rib, but that a chronic aseptic inflammatory lesion in the nerves immediately related to the rib is produced by the pressure irritation. This lesion, of course, would only clear up gradually on removal of the exciting cause. In this connection the thickening of the endoneurium and the proliferation of endoneurial nuclei observed by us in the lower trunk of the brachial plexus (particularly in its inferior part) and in the distal part of the first dorsal nerve where it came into immediate relation with the cervical rib, appear to us to be of special significance. We interpret these histological findings as evidence of irritation by the rib in the immediately related nerves.

As stated above, the collection of nerve-fibres (largely unmyelinated) which, for a distance of $\frac{1}{2}$ in., had a separate existence in the inferior part of the lower trunk of the plexus, cannot be considered as an unjoined ramus as Telford and Stopford claim for the separate bundle of unmyelinated fibres described by them. In the present specimen the number of unmyelinated fibres in this collection forms only an extremely small fraction of the total number of unmyelinated fibres in the lower trunk of the plexus, and it is difficult to believe that irritation of these few unmyelinated fibres can be responsible for such widespread arterial spasm as seen in the case reported. Indeed, the much more numerous unmyelinated fibres in the inferior parts of the adjacent fasciculi of the lower trunk of the plexus must also be exposed to the effects of pressure by the rib, and the histological evidence supports this contention. The fact that only the lower trunk and the first dorsal nerve showed histological evidence of proliferation of endoneurium and endoneurial nuclei, and our interpretation that this lesion was responsible for the widespread irritative effects on the sympathetic vasoconstrictor fibres to the limb vessels, implies that a large proportion of the vasoconstrictor unmyelinated fibres enter the limb by way of the lower trunk, or that these fibres in the lower trunk of the plexus have a particularly widespread distribution to the arteries of the limb. We have already pointed out that unmyelinated fibres in general are more numerous in the lower trunk.

In view of the work of Ranson and Davenport,⁵ supported by Donal Sheehan,⁶ not all the unmyelinated fibres in the trunk must be considered to be efferent sympathetic fibres, these authors believe that many unmyelinated fibres of afferent protopathic function pass via the posterior nerve-roots to small cells in the posterior root ganglia.

As the material was not to hand for examination, we are unable to give any information as to whether the nerves on the under aspect of the subclavian artery, derived from the sympathetic chain and from the ansa subclavia (Hovelacque?), were in any way affected.

The lesion in the case under discussion has evidently affected principally the unmyelinated vasoconstrictor fibres in the inferior part of the plexus. It is possible that different degrees of pressure-induced chronic inflammation affect the different types of nerve-fibres in the plexus to a variable extent, hence the different clinical types of cases of cervical rib. At any rate, in our case there is no *anatomical* segregation of the unmyelinated fibres which would explain their special involvement.

If the chronic inflammatory change progresses to permanent fibrosis, this would account for the failure to relieve symptoms which is known to occur in some cases of operation for cervical rib. It therefore seems desirable that operative treatment, whether removal of the cervical rib or division of the scalenus anterior muscle allowing the rib to droop, should be undertaken as early as possible to relieve the plexus from harmful pressure.

SUMMARY AND CONCLUSIONS

1 The brachial plexus from a case of cervical rib with vascular symptoms was examined histologically in serial transverse section.

2 All grey rami joining the component roots of the lower trunk of the plexus had become uniformly distributed within the roots and trunk proximal to the region in contact with the cervical rib.

3 Marked endoneurial thickening with nuclear proliferation was seen in the first dorsal nerve and inferior part of the lower trunk of the plexus where they were related to the cervical rib.

4 It is suggested that this lesion is responsible for the irritation of the sympathetic fibres producing the widespread vasoconstriction in the limb.

REFERENCES

- ¹ TELFORD, E. D., and STOPFORD, J. S. B., *Brit Jour Surg*, 1931, viii, 557.
- ² BLAIR, D. M., and DAVIES, F., *Lancet*, 1933, i, 1113.
- ³ BLAIR, D. M., and DAVIES, F., *Ibid*, ii, 801.
- ⁴ WOOLLARD, H. H., *Jour of Anat*, 1931, lvi, 147.
- ⁵ RANSON, S. W., and DAVENPORT, H. K., *Amer Jour Anat*, 1931, xliii, 331.
- ⁶ SHEEHAN, D., *Anat Record*, 1933, lv, 111.
- HOVELACQUE, A., *Ann d'Anat pathol*, 1929, vi, 968.

DIFFUSE INTRADUCT CARCINOMA OF THE BREAST

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SEVERAL considerations have stimulated us to report these eleven cases of diffuse intraduct carcinoma of the breast. The disease is rare—according to Cheate and Cutler, the rarest of all forms of cancer of the breast—and yet it occurs frequently enough in any long series of cases to have a definite practical importance for the surgeon. Its clinical features differ so widely from the conception of breast cancer by which the surgeon is commonly guided that it is perfectly easy for the diagnosis to be missed, even after macroscopic examination of the amputated breast. The point we would particularly emphasize is that the breast affected with diffuse intraduct carcinoma does not contain a localized nodule of growth infiltrating the stroma, but is the site of a widespread malignant change of the epithelium of its ducts and acini affecting one-third to two-thirds of its substance. The naked-eye appearances, characteristic as they are when correlated with the microscopic picture, are so different from those of ordinary breast cancer that the pathologist who has not seen a case may easily fail to recognize that he is dealing with a malignant condition. The simulation of polycystic disease may be very close. Intraduct carcinoma is one of the conditions in which the surgeon is likely to be in doubt as to procedure, and to call for guidance from the pathologist during the course of the operation. Before it is possible to give this guidance, it may be necessary for frozen sections to be cut. The histological picture presented by the sections is most unusual. It strongly suggests that the disease starts simultaneously throughout a number of ducts and their acini, and that it does not spread from a primary focus.

So far intraduct carcinoma has most often been described in association with Paget's disease of the nipple, a condition in which it is always present in some degree. Ewing has described it apart from Paget's disease, under the term 'comedo-carcinoma'—a reference to the peculiar naked-eye appearance of the sections. Cheate and Cutler¹ have met with 8 cases of the diffuse disease, 2 of which were in breasts also the site of Paget's disease. They consider it a highly malignant condition. 6 of their 8 patients died within four years of first coming under observation.

CLINICAL CONSIDERATIONS IN ELEVEN CASES

The average age of the 11 cases here described was 43, the oldest was 61 years and the youngest 32, 7 of the patients were married and 4 unmarried. Five had had children, and possibly a sixth in whose case the history of pregnancy is omitted from the clinical notes. The question of lactation is mentioned in 2 cases, in both of which it was abnormal. One patient was able to suckle her two children

for one month only, and the other patient, who had six children, discontinued suckling on account of pain with each of the last two babies. We have considered these particulars in some detail in order to compare them with the history of cases of Paget's disease of the nipple as we have been able to obtain this from the



FIG 274—Photograph of cut surface of large breast containing diffuse intraduct carcinoma. Shows dilated ducts and affected alveoli standing out from surrounding stroma (Unmagnified)



FIG 275—Section of same breast as Fig 274 (unmagnified) showing the diffuse nature of the change

literature. The average age of patients with Paget's disease when they first come under observation seems to be rather higher. Cheate and Cutler reported 17 cases in which the average age was 53. A more striking contrast is found in the fact that of 37 women with Paget's disease which we have collected from the

literature, 34 were married, whereas of our 11 patients, 4 were unmarried. The two cases in our series with evidence of Paget's disease both occurred in married women.

The symptom for which these patients sought advice was either the presence of a lump in the breast or the occurrence of discharge from the nipple. The length of the history was comparatively short, in 1 case only was it over a year, and in 7 cases it was considerably less, varying from one to five months. As we have noticed in other diseases of the breast, discharge from the nipple, particularly if it is blood-stained, brings the patient to the doctor earlier than does the detection of a lump. In these cases of diffuse intraduct carcinoma, discharge was present in 7 of the 11. The discharge was blood-stained in 3 cases, serous in 3, and purulent in 1. The frequency of discharge from the nipple is particularly note-



FIG 276 —High-power view of section shown in Fig 275. Unaffected breast at A, and affected alveoli at B.

worthy in view of the tendency to regard this symptom as more or less pathognomonic of simple, non-malignant duct papilloma.

Careful descriptions of the condition of the affected breasts are available in almost every case. These show that the affected breast was definitely larger than the other in 4 cases, and œdematous in 3. In 2 cases the presence of *peau d'orange* is noted, 3 of the breasts are described as 'red and shiny', and 1 of these was also said to be 'hot'. Palpation of the breast in every case except one revealed the presence of a lump, of which the patient was in some cases aware. The lump was usually quite ill-defined, and is variously described as 'indefinite', 'irregular', 'a mass of lumps', 'a diffuse hard area', 'a craggy mass'. It is frequently stated that the whole breast was œdematous, or that the breast was 'all rather hard'. Definite hard glands were present in 3 cases only. In 6 cases it is specifically stated that no glands were palpable. The nipple was retracted

in 4 cases, in 1 it was the site of Paget's disease, and this was, incidentally, the breast in which no lump was to be felt, and the case showing the least extensive growth of the series

Personal knowledge of the last case of the series leaves us in no doubt that clinically the condition can be very puzzling. The breast was large, heavy, and œdematous, *peau d'orange* was well-marked, and over the upper inner quadrant there was a patch of bluish discoloration of the skin. An indefinite lumpiness was to be felt with no defined edge. The nipple was normal. A local excision was done, taking a wide margin of skin, and the breast sent at once to the pathological laboratory. Even after careful naked-eye examination of the cut surface the condition looked innocent, so the operation was completed without clearing the axilla. Later microscopical examination showed a most active malignant growth of the ducts, with widespread lymphatic permeation. The axillary glands were then excised and were found to be infiltrated.

MACROSCOPIC APPEARANCES (*Figs 274, 277*)

The appearances depend on the size of the breast, and a comparison of the effect of the disease in the large fat type of breast with that in the small, rather atrophic breast largely explains the variety of the clinical descriptions. In the first place, it cannot be too well emphasized that there is no well-defined mass of carcinoma spreading into the surrounding tissue. A large breast containing much glandular tissue and a large amount of fat will be of increased consistency throughout. The cut section shows the ducts thickened and dilated, often distended with yellow secretion. The alveolar tissue looks opaque and stands out against the background of fat. The general appearances of the section reproduce the normal breast pattern, but in an exaggerated degree.

The section of a small breast with a small amount of glandular tissue and very little fat is rather different. It presents a honeycomb appearance owing to the presence of numerous small cysts formed by the dilated and distended ducts. The presence of yellow nodules of growth in some of the ducts gives an appearance to the section which should suggest duct carcinoma, and it was these nodules that gave rise to the term 'comedo-carcinoma' in Ewing's description. Certain cases of Reclus' disease present a somewhat similar picture, where the secretion in the cysts has become opaque and simulates nodules of growth. In some cases of polycystic disease it is impossible to exclude malignancy without microscopical examination, but, whereas in diffuse intraduct carcinoma there is, in our experience, always some unchanged breast tissue to be observed, in polycystic disease the change is present throughout the breast tissue. The secretion in intraduct carcinoma may be blood-stained, or there may be a golden or brown pigmentation of the connective tissue, due to absorption of hæmoglobin. This appearance has not been observed by us in simple cystic disease.

Serial sections of the breast, 2 mm thick, may reveal small areas where the cystic formation is less marked and in which the tissue seems firmer than elsewhere. These areas, examined microscopically, show infiltration by growth of the connective tissue, but such areas are not very conspicuous, and are likely to be missed unless the breast is systematically examined. The axillary glands may be enlarged and firm and may contain obvious growth.

MICROSCOPICAL APPEARANCES (*Figs 276, 280-282*)

The histological picture varies with the part selected for section. If the section is made of only a small part, it will show what appear to be dilated ducts

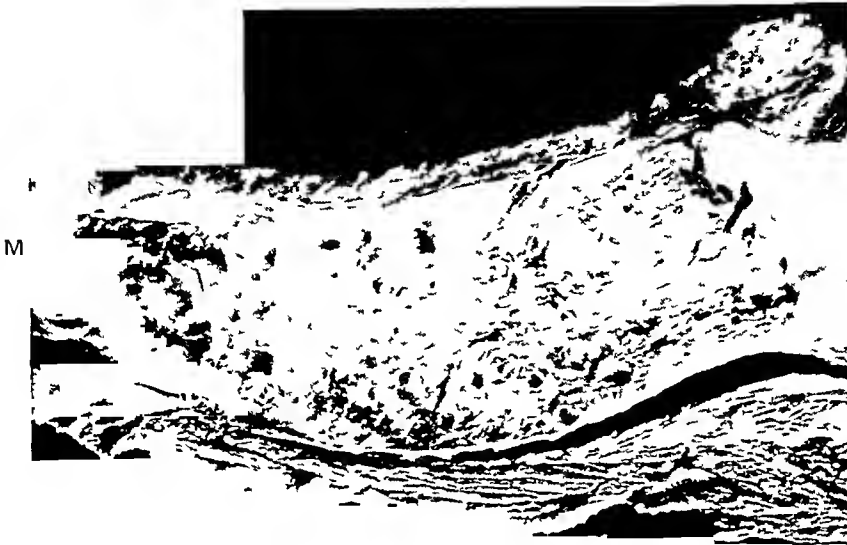


FIG 277 —Photograph of cut surface of small breast, with diffuse intraduct carcinoma extending into the fat at the margin, M (Slightly magnified)



FIG 278 —Diffuse cystic disease of the breast. Compare the similarity of the macroscopic appearance to that of Fig 277 (Unmagnified photograph of section)

containing malignant-looking epithelium, but no alveoli may be seen and no infiltration outside the duct wall. Whole section of the breast—or, where this is impracticable, of half the breast—is most helpful, both in determining the condition

of the acini and in tracing the infiltration of the stroma. Staining the elastic tissue throws light on the structure of the growth. Tubules of epithelium will be seen closely resembling the ducts with their proliferated epithelial lining, except that the elastic coat is missing (*Fig 282*). Some of these outgrowths are alveoli, dilated



FIG 279 —Section of same breast as *Fig 278*. Microscopical examination showed no evidence of malignancy (Unmagnified)

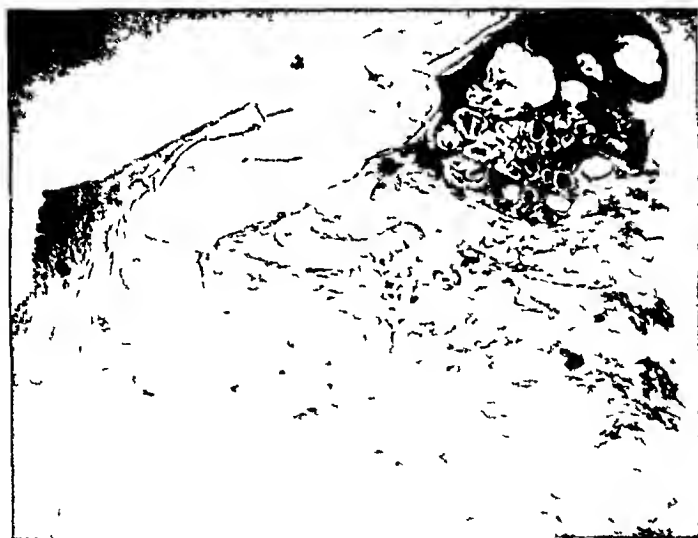


FIG 280 —Unmagnified photograph of stained section of breast with diffuse intraduct carcinoma for comparison with *Fig 279*. In this case there was widespread lymphatic permeation

and undergoing malignant transformation. In large sections alveoli can be detected at the edge of the growth in various stages of change—an appearance that is most easily demonstrated in the larger breasts (*Fig 276*). In other parts of the section the 'tubules' will be found to represent outgrowths of secondary infiltration of the epithelial lining of the duct. So closely do they resemble the ducts from

which they arise that at first one finds it difficult to convince oneself that their appearance is not accounted for by a fault in the staining technique. However, by careful search an outgrowth may be found actually breaking through the elastica of the parent duct (Fig 282, A)



FIG 281 —Section of small breast showing the dilated ducts containing growth and infiltration of the stroma at X ($\times 3$)



FIG 282 —High-power view of section showing invasion of the stroma. A, Growth breaking through the elastic tissue, B, Tubules of secondary growth in stroma. Inflammatory reaction well seen.

In 9 of our cases infiltration of the stroma could be detected in one or more areas. To find these areas it was sometimes necessary to make microscopic sections of the whole breast, in other cases slicing of the whole breast, followed by section of likely-looking parts, was successful. In all cases there is a more or

less well-marked inflammatory reaction in the stroma, plasma cells and lymphocytes predominating. So striking is the occurrence of the carcinomatous change in such a widespread area that the conclusion is almost irresistible that there has been a simultaneous affection of the ducts and acini throughout the greater part of the breast. From this conclusion the suggestion arises of a blood-borne stimulus, possibly of a hormonal nature.

OPERATION AND POST-OPERATIVE HISTORY

That the condition of the breast was not always recognized to be a malignant one is suggested by the fact that radical amputation was performed in only 6 cases. The remaining 5 patients were treated by local amputation of the breast and excision of the axillary lymph-glands. In one patient the dissection of the axilla was done after an interval of three months, when recurrence was obvious.

We have been able to trace the after-history of 7 of these 11 patients. Of the 6 treated by radical amputation, 2 were untraced, and 1 was lost sight of after one year. Three are living at periods of seven to eight years after operation, but one of these three shows evidence of recurrence in the chest wall and the lung.

One of the 5 cases treated by local excision of the glands and breast is alive and well twelve years after, another shows no evidence of recurrence two and a half years after operation. The most recent case was operated on nine months ago, and proved to be a very advanced case. She is so far free of recurrence.* One patient is definitely known to have died of secondary growth eighteen months after operation, and the fifth case we have been unable to trace.

RELATION OF INTRADUCT CARCINOMA TO PAGET'S DISEASE OF NIPPLE

Muir² has brought forward striking evidence to support the view, first advanced by Jacobaeus³ in 1904, that the primary lesion in Paget's disease is a malignant epithelial hyperplasia within the ducts. The lesion of the nipple he believes to be due to a direct intradermal spread of carcinoma cells from the ducts, and the deep-seated infiltration in the underlying breast tissue, with which Paget's disease is so often associated, to be an outgrowth from the deeper parts of the ducts at a place where the epithelial change is more actively malignant. In 5 cases Muir was able to demonstrate an undoubted association of duct carcinoma with Paget's disease, though in 2 cases Paget's disease was discovered only on microscopic examination. Cheate and Cutler have described 17 cases of Paget's disease all associated with intraduct carcinoma, and 6 cases of diffuse intraduct carcinoma apart from Paget's disease. Two of our cases showed evidence of Paget's disease, one on microscopic examination only.

We have sufficient evidence in the sections of our cases to convince us that infiltration of the lymphatics⁴ can have no bearing on the causation of Paget's disease. The last case showed the most marked infiltration of the subdermal tissue, extending right under the skin around the nipple, but no infiltration of the epidermis itself, and no evidence of Paget's disease.

* When last seen (14 months after operation) this patient showed extensive infiltration of the skin of the chest wall.

Muir describes the epithelium confined within the ducts as being in a state of potential malignancy. The history of patients with the disease suggests that they are suffering from a much less malignant type of growth than are those with diffuse intraduct carcinoma. Our series of 11 cases seems to be intermediate between the highly malignant growths described by Cheate and Cutler and the comparatively mildly malignant epithelial change found in Paget's disease. We conclude that in all these clinical types of growth one is dealing essentially with the same disease, which when slowly developing allows of permeation of the skin around the nipple, and when rapidly spreading leads to infiltration of the lymphatics and to widespread growth at an early stage of its history.

SUMMARY

1 Eleven cases are reported of diffuse intraduct carcinoma of the breast. The clinical history and progress of these cases are described.

2 The change is diffuse and affects both ducts and acini. It should be considered a malignant condition because infiltration of the stroma, lymphatic permeation, and outgrowth of the epithelium of the ducts can be demonstrated.

3 The widespread nature of the change and its uniformity throughout the breast suggest the action of a blood-borne stimulus, possibly of a hormonal nature.

4 This series of cases illustrates all types of intraduct carcinoma, from that of low malignancy and slow growth with which Paget's disease tends to be associated, to the highly malignant type in which lymphatic permeation is a marked feature.

The photographs illustrating the text were taken by the technician to the Pathological Department, C. Wells.

REFERENCES

- ¹ CHEATLE and CUTLER, *Tumours of the Breast*, 1931.
- ² MUIR, R., *Four Pathol and Bacteriol*, 1927, xxx, 451.
- ³ JACOBÆUS, H. C., *Virchow's Arch*, 1904, clxxviii, 124.
- ⁴ HANDLEY, W. SAMPSON, *Brit Jour Surg*, 1919-20, vii, 183.

SERIAL RADIOGRAPHIC APPEARANCES OF A NEUROPATHIC SHOULDER-JOINT

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THE details and serial radiographs of the following case illustrate the different phases which were found in a neuropathic joint during its progress, and suggest that the classification of such joints into (1) hydro-arthritis, (2) atrophic, (3) hypertrophic types may merely represent these different phases of the lesion

Mrs W, aged 52 years, consulted Mr Bertram Lloyd at the Queen's Hospital in August, 1931, complaining of stiffness and swelling of the left shoulder. The condition had arisen insidiously during the past few months. No history of definite trauma could be obtained. On examination the shoulder was found to be swollen and painful. The radiograph taken on Aug 18, 1931, (*Fig 283*) shows a marked swelling of the soft tissues around the shoulder-joint. No definite bone changes can be detected beyond a slight suspicion of increased depth of the glenoid cavity. No abnormal shadows are to be seen in the joint. The appearances are those of a hydro-arthritis. Aspiration was performed, and following this ionization was given for three months. Slight improvement was recorded as the result, and as the condition appeared to be non-progressive, treatment was discontinued.

In March, 1933, pain caused the patient to seek further hospital treatment, and on examination it was found that the condition had progressed and the swelling had increased. The patient was admitted to the Nerve Hospital under Dr Geoffrey Eden for investigation. A radiograph taken on March 14, 1933, (*Fig 284*) shows (1) that the swelling of the soft tissues had increased, (2) that the head of the humerus and the walls of the glenoid had been worn away as if the bone had been rubbed down with a file, (3) that a large collection of amorphous calcium had been stored up within the joint capsule, and (4) that the affected bones are normal in density. These appearances are typical of the so-called atrophic neuropathy.

A further examination of the patient was made on Nov 27, 1933, when she reported that though the shoulder was troublesome she was able to perform her household duties satisfactorily. The joint area was swollen considerably and ached. The swelling and discomfort appeared to vary, being much more noticeable some days than others. No disability, discomfort, or sensory changes had been noted by the patient in any other region with the exception of the left hand, which always appeared to be cold.

On examination the uniform and regular swelling of the left shoulder-joint area was obvious. When the arm was raised above the shoulder level the movements were restricted, but there was definite abnormal forward mobility of the upper end of the humerus. The covering deltoid appeared to be hypotonic, but the area did not pit on pressure. The fullness and fluctuation of the enlarged



FIG 283 —Radiograph of the left shoulder showing the swelling over the shoulder-joint and a suspicion of increase in the depth of the glenoid but no other bone abnormality (Aug 18, 1931)



FIG 284 —Radiograph showing that the upper end of the humerus and the glenoid fossa have been worn away. A large collection of amorphous calcium is shown in the joint, and there is a marked increase in the swelling of the soft tissues over the joint. It will be noted that the bone destruction is not associated with any change in the density of the affected bones (March 14, 1933)

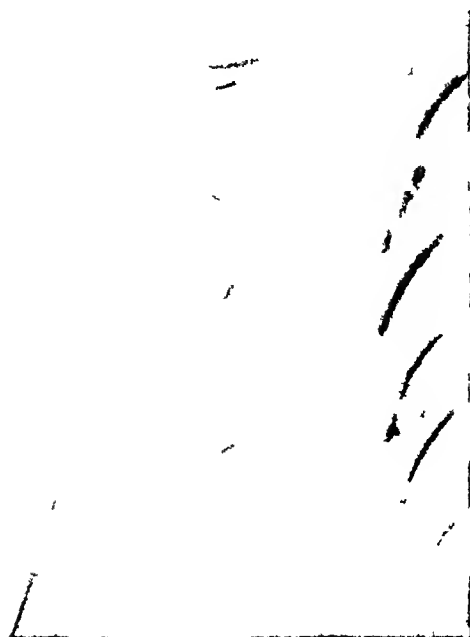


FIG 285—Radiograph showing a decrease in the size of the swelling over the joint, a massive bony shelf built on to the glenoid, and disappearance of the amorphous calcium from the joint capsule (Nov 27, 1933)



FIG 286—Radiograph showing that the new glenoid shelf has undergone some absorption (Feb 10, 1934)

joint could be detected in the axilla. Fluctuation could also be elicited in the region of the delto-pectoral groove. The left arm appeared to be half an inch shorter than the right. No very marked changes could be detected in the central nervous system. The pupils were unequal and slightly irregular, the reactions sluggish, but the ocular movements were full. There was anæsthesia of the posterior pharyngeal wall, absence of the palatal reflex, and a slight tremor of the tongue. No other definite abnormality could be detected. The cerebrospinal fluid showed a normal cell content, the Wassermann reaction was negative, and the biochemical tests were normal.

An X-ray taken on Nov 27, 1933, (*Fig 285*) showed a very marked alteration in the radiographic appearances. It will be seen that the upper end of the humerus has been worn away still more and the large deposit of amorphous calcium has disappeared from the joint cavity, but a massive bony shelf has been built on to the glenoid of the scapula, the swelling of the soft tissues not being so marked as at the previous examination. These appearances now suggest a hypertrophic arthropathy.

On Feb 10, 1934, a further examination was made which revealed similar clinical features to those recorded at the previous examination, but the radiograph (*Fig 286*) taken on this date showed that some absorption of the enlarged glenoid had occurred.

The destruction of bone and the storage within a joint of amorphous calcium set free in the process, and its subsequent use in the growth of bone, is a very interesting feature of the case.

The writer has previously suggested¹ that in osteochondritis deformans juvenilis coxæ (Legg-Perthes' disease) such calcium interchange may take place in the bone associated with the contents of the capsule of a joint, for in this condition we see from a study of serial radiographs that in the early phases the epiphysis takes up calcium and becomes denser, while the end of the diaphysis loses calcium and becomes radiotranslucent. Gradually the epiphysis loses its calcium, and concomitantly the end of the diaphysis approaches the density of normal bone. Eventually stability is reached when the epiphysis and diaphysis have the density and structure of normal bone.

The illustrations used in this article are reproductions made by printing through the original radiograph and a contact positive film taken from the latter. The two films, the original and its positive, are so arranged in the printing frame that the two images are not quite superimposed when the printing paper is fixed in position. The advantages of the prints obtained in this way are that the details of the dense bony structures can be preserved along with those of the outlines of the soft tissues, and a relief picture of the structures is so obtained, whereas with the ordinary straight print from a radiograph the details of the soft tissues, or of the bones, are lost because of the marked contrast in the densities.

My thanks are due to Mr Lloyd and Dr Eden for permission to publish this case.

REFERENCE

- ¹ BRAILSFORD, JAMES F, *The Radiology of Bones and Joints* London J & A Churchill

RENAL-ADRENAL ADHERENCE

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DEVELOPMENTAL and other non-pathological abnormalities of organs at times assume considerable clinical importance, particularly to the surgeon. Variations of this nature in the adrenals are not often met with, and as the total number of operative procedures concerned with these glands is very small, there is little call for the surgeon to take note of minor variations in their size, shape, position, or vascular supply. Furthermore, the great majority of operations on the adrenals aim either at complete excision of the organ or, more rarely, at the removal from it of tumours such as the rare pheochromocytoma, or chromaffinoma. In view, however, of the vital importance of these organs it is obviously a matter of the greatest concern that in the numerous operations nowadays performed upon the kidneys every possible precaution should be taken to prevent serious damage to the adrenals. A perusal of the text-books of surgery in general use will reveal that, whereas great stress is always laid upon the determination of the existence of a functioning kidney on the unaffected side prior to the undertaking of a nephrectomy, the question of the possible interference with the function of the adrenals by such operative procedures receives little or no attention. This is in all probability due to one or other of three reasons: (1) In the great majority of cases nephrectomy is effected without any damage to the adjacent adrenal capsule, (2) No recognized pre-operative method exists for determining the presence of adrenal abnormalities, (3) The existence of a not uncommon abnormality in the renal-adrenal relationship which renders serious damage to the adrenal inevitable in the course of a nephrectomy is not sufficiently appreciated.

In the course of the routine performance of approximately 1500 post-mortem examinations I have on 6 occasions met with a degree of bilateral renal-adrenal adherence which would have rendered impossible the removal of the kidney without the coincident removal, or serious laceration, of the adrenal gland. The accompanying photographs (*Figs 287, 288*) show this condition in the case of a young woman of 22 years. In all these cases the small amount of loose connective and fatty tissue which usually separates the antero-superior surface of the kidney from the postero-inferior aspect of the superposed adrenal gland was absent in whole or part, and in no case could a nephrectomy have been performed without coincident adrenalectomy. In some of the cases the lower portion of the adrenal was thinned out and applied closely to the upper third of the anterior surface of the kidney, and section showed either complete absence of any capsular connective tissue between the two organs, or else the presence of only a single attenuated layer of fibrous tissue separating the two organs and serving as a capsule for both. The accompanying microphotographs illustrate the two degrees of adherence referred to (*Figs 289, 290*).

In some of the cases the renal capsule appears to be continuous with that of

the adrenal, i.e., the two organs appear to be enclosed in a single fibrous-tissue capsule. This is always more marked on the anterior aspect, and usually the continuity of capsule is localized to the central portion of the inferior border of the adrenal gland. Even in the most pronounced of these cases there is always a small interspace filled with connective tissue between the infero-mesial angle of the adrenal and the kidney. In the cases where the degree of adherence is less marked, the separate capsules of the two organs are fused over the greater part of their surfaces of contact, even though the whole of the inferior border of the adrenal appears to be free and capable of being raised slightly from the underlying kidney.

The cause of this condition is readily appreciated from a study of the development of the adrenals. The adrenal cortex is developed from the mesial aspect of the upper, or cephalad, end of the Wolffian ridge, from the lower end of which



FIG. 287.—Renal-adrenal adherence. Right kidney showing marked adherence along the middle portion of the inferior border of the adrenal.



FIG. 288.—Renal-adrenal adherence. Left kidney. The incision shown about one-third up from the inferior border of the adrenal was made for the purpose of microscopical section.

is developed the testis or ovary, while the kidney lobules are developed from a mass of undifferentiated cells lying some distance below this. When, about the second month of intra-uterine life, the gonads migrate downwards, the adrenal cortex approximates itself to the kidney. At this stage the foetal adrenal cortex is considerably larger than the kidney and it envelops the upper pole of the collection of renal lobules. Normally the further development of these two structures proceeds independently, each becoming enclosed in its own distinct fibrous tissue capsule with the intervention of a small amount of fibro-fatty tissue. It can, however, readily be appreciated that the close approximation of the two organs in the second and third months of intra-uterine development may at times result in a more intimate relationship, which may persist into adult life. On this hypothesis is based the explanation of the presence of adrenal 'rests' in the kidney, and the acknowledged presence of these provided the anatomical basis for Grawitz's



FIG 289 —Renal-adrenal adherence Section showing complete absence of capsule or inter glandular tissue ($\times 50$)



FIG 290 —Renal-adrenal adherence Section showing single thin fibrous capsule serving for both organs ($\times 80$)

theory of the origin of the hypernephromata These 'rests' are usually of small size and are found in various situations within the kidney, though mainly sub-capsularly or interlobularly in the upper pole That the persistence into adult life of the fœtal condition of intimate relationship may be on a much larger scale, though more superficial, is well illustrated in the cases under discussion

A careful examination was made in all these cases for evidences of other forms of developmental abnormalities, with completely negative results In particular, in none were there macroscopic evidences of 'adrenal rests' within the renal parenchyma, nor were any of these 'rests' discovered in the considerable number of sections cut for microscopical examination As all the 6 cases referred to were adults, it is not surprising that in none of these were found any ectopic adrenal fragments, but in view of the frequent occurrence of such scattered nodules of adrenal cortical tissue—especially on and near the liver and in the pelvis or along the round ligament or spermatic cord—in children, it would be of interest to know whether adrenal nodules of this type are more frequent in children with renal-adrenal adhesions than in those in whom the normal relationship exists between the kidney and the adrenal gland

As stated above, there is at present no way of foretelling the existence of this condition, and it follows that nephrectomy in these cases inevitably leaves the patient with only one intact adrenal gland The unintentional removal or laceration of an adrenal gland under these circumstances will probably result in considerable hæmorrhage, and thus add to the dangers of the operation The intimate connection of the adrenal medulla with the solar and celiac plexuses of sympathetic ganglia and nerves must surely imply a severe degree of shock when these nervous connections are rudely severed, and it is just possible that some of the otherwise inexplicable early deaths following nephrectomy by competent surgeons may be the result of this The condition of renal-adrenal adherence is apparently always bilateral, and any subsequent operation on the remaining kidney is thus fraught with the gravest of dangers Fortunately the surgeon will but rarely be faced with this danger, but there is one condition in which it should be borne in mind—namely, in nephrolithotomy performed on a single remaining kidney The usual operation for renal calculus, including, as it does, the delivery of the kidney on to the surface through a lumbar incision, would, in the presence of renal-adrenal adherence, result in laceration of the single remaining adrenal with serious or fatal result Such a result could be obviated only by avoiding the manœuvre of delivering the kidney from its bed, and whereas it is obviously incumbent upon the surgeon to take this special precaution in cases of nephrolithotomy in which it is *known* that earlier nephrectomy resulted in coincident adrenalectomy, it is a matter for serious consideration whether this should not be recommended as the procedure to be adopted in *all* cases of nephrolithotomy performed on patients possessing only one kidney In the absence of definite knowledge of the condition of the adrenal on the side of the old nephrectomy it can hardly be regarded as justifiable to presume that it is intact, particularly in view of the fact that the lumbar route of approach to the kidney exposes first the posterior surface and lateral border of the kidney, from which nothing at all can be learned as to the possibility of the existence of an adherent adrenal gland Only when the kidney has been withdrawn from the depths of the wound to or near the surface does such a condition become apparent, by which time severe damage may have been done

In discussing this matter with the surgeons of the hospitals from which the post-mortem experience was gained, the impression was gathered that when in the past portions of adrenal tissue were occasionally found attached to kidneys removed surgically, the operations performed had been regarded as suspect—that is to say, the surgeons felt that probably they had been a bit careless in the freeing of the kidney from its bed. One of these surgeons has recently made a careful examination of the kidneys surgically removed and has twice met with the condition of renal-adrenal adhesion. In one case the complete adrenal gland was removed intact with the kidney, in the other case a large portion of the gland was found adherent to the kidney. These facts have been emphasized in the hospital case sheets of the patients, who have been instructed that owing to an abnormal arrangement of their organs it is most important that their hospital case sheets should be consulted before any further operation is performed upon their single remaining kidney. That such subsequent operations are occasionally necessary must have been the experience of most surgeons working in the field of urology, and even though, by the law of chance, the possible occasions on which the condition of renal-adrenal adherence will complicate operations of this type is very small, the issues at stake are too high to justify neglect of the precautions by which an almost inevitably fatal accident might arise.

I have much pleasure in acknowledging my indebtedness to Dr H. H. MacWilliam and Mr Kirk Wilson of Walton Hospital for permission to make use of the material on which this communication is based, and to Mr Wilson for his co-operation in the matter of the search for evidence of damage to the adrenals in all cases of nephrectomy at the hospital.

COLLECTIVE INQUIRY BY THE FELLOWS OF THE ASSOCIATION OF SURGEONS INTO GASTROJEJUNAL ULCERATION

BY GARNETT WRIGHT

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TWENTY-FIVE years ago in his classic paper, "Jejunal and Gastrojejunal Ulceration following Gastrojejunostomy", Paterson¹ said that the fear of the occurrence of this condition cast a faint shadow over the otherwise admirable results of the operation, and he expressed the opinion that the incidence was under 2 per cent and that there were signs that this was diminishing. With the passing years the shadow has grown more dense. Increasing recognition of the symptoms, and perhaps a keener and more persistent investigation of unsatisfactory results, have resulted in a gradual rise in the estimated risk of post-operative ulcer.

Paterson distinguished two kinds of these ulcers: (1) Jejunal—situated in the jejunum, and (2) Gastrojejunal—situated on the anastomosis and extending to the gastric or jejunal mucosa, or both. The latter term has been extended by the profession at large to include all forms of post-operative ulcer. I would suggest that in order to avoid confusion the term 'secondary peptic ulcer' be used as a general term which would cover all varieties of post-operative ulcer. Individual ulcers should be called 'jejunal', 'anastomotic', or 'gastric', according to their exact situation. Some, indeed, may be duodenal, as after gastroduodenostomy.

INCIDENCE OF SECONDARY ULCERATION

Very wide estimates of the incidence of this condition have been made, from under 2 per cent by Paterson to over 20 per cent from one New York clinic. One reason for this difference is lack of uniformity in the method of arriving at the estimate. It is obviously misleading to make it from an analysis of all cases of gastrojejunostomy, irrespective of the lesion for which the operation was done. It is generally agreed that gastro-enterostomy for duodenal ulcer is the operation most likely to be followed by secondary ulceration.

In the Collective Inquiry by the Fellows of the Association of Surgeons of Great Britain and Ireland the various operations have been analysed in relation to the three main gastric lesions, viz, duodenal ulcer, gastric ulcer, and carcinoma. In this way it is possible to arrive at some reasonable estimate of the risks in various operations for different lesions. These have been analysed in *Table I*, and it is important to note that the cases here considered are not necessarily the same as those in the analysis of individual ulcers. *Table I* covers a period of operations done in the five years ending 1930, while the individual cases of ulcer were collected over a period of ten years, 1923-32 inclusive. This accounts for certain minor differences between the two.

Table 1—INCIDENCE OF GASTROJEJUNAL ULCERATION FOLLOWING VARIOUS OPERATIONS

	MALES	FEMALES	TRACED	GASTROJEJUNAL ULCERATION		PERCENTAGE OF COMBINED PROVED AND SUSPECTED CASES
				Proved by Operation	Diagnosed by Symptoms	
Gastrojejunostomy *						
1 POSTERIOR GASTROJEJUNOSTOMY						
For duodenal ulcer	2310	424	1730	70 (4.04%)	77	8.49
For gastric ulcer	670	214	507	27 (5.32%)	26	10.45
For gastric carcinoma	151	69	140	0	0	—
With excision of gastric ulcer	84	28	105	0	0	—
With excision of duodenal ulcer	2	0	2	0	0	—
With excision of gastric ulcer and duodenal ulcer	1	1	1	0	0	—
With cautery of gastric ulcer	19	5	24	0	0	—
2 POSTERIOR GASTROJEJUNOSTOMY WITH ENTERO-ANASTOMOSIS						
For duodenal ulcer	48	9	33	1 (3.3%)	1	6.6
For gastric ulcer	29	20	28	0	0	—
For gastric carcinoma	8	6	5	0	0	—
3 ANTERIOR GASTROJEJUNOSTOMY						
For duodenal ulcer	103	13	85	6 (7.06%)	2	9.4
For gastric ulcer	74	41	85	1 (1.2%)	0	—
For gastric carcinoma	49	25	47	0	0	—
With excision of gastric ulcer	1	2	3	0	0	—
With cautery of gastric ulcer	3	0	3	0	0	—
4 ANTERIOR GASTROJEJUNOSTOMY WITH ENTERO-ANASTOMOSIS						
For duodenal ulcer	42	4	25	6 (24.0%)	1	28.0
For gastric ulcer	21	7	23	1 (4.34%)	0	—
For gastric carcinoma	32	12	31	0	0	—
5 PYLORODUODENOSTOMY	1	1	1	0	0	—
Gastroduodenostomy for duo- denal ulcer	34	5	29	1 (3.45%)	0	3.45
Partial Gastrectomy						
1 ANTERIOR POLYA						
For duodenal ulcer	34	8	29	0	0	—
For gastric ulcer	178	116	199	1 (0.5%)	1	1.0
For gastric carcinoma	79	48	93	0	0	—
2 ANTERIOR POLYA WITH ENTERO- ANASTOMOSIS						
For duodenal ulcer	2	0	1	0	0	—
For gastric ulcer	1	3	2	1	0	—
For gastric carcinoma	1	2	3	0	0	—
3 POSTERIOR POLYA						
For duodenal ulcer	81	25	77	0	2 X-ray	2.59
For gastric ulcer	267	167	306	0	1	0.3
For gastric carcinoma	70	31	80	0	0	—
Gastric ulcer and duodenal ulcer	14	9	18	0	0	—
4 POSTERIOR POLYA WITH ENTERO- ANASTOMOSIS						
For duodenal ulcer	0	0	0	0	0	—
For gastric ulcer	0	1	1	0	0	—
For gastric carcinoma	0	0	0	0	0	—
5 BILLROTH No I				Gastroduodenal ulceration		
For duodenal ulcer	43	12	38	0	8	21.0
For gastric ulcer	80	37	85	2 (2.4%)	1	3.5
For gastric carcinoma	26	6	23	0	0	—
6 OLD TYPE BILLROTH, No II				Gastrojeunal ulceration		
For duodenal ulcer	3	0	1	1	0	—
For gastric ulcer	15	5	11	0	1	—
For gastric carcinoma	15	13	14	0	0	—

* With or without procedures on actual lesions (excision, infolding, cauterization, etc.)

SUMMARY INCIDENCE OF GASTROJEJUNAL ULCER AFTER ALL CASES OF OPERATION *

For Duodenal Ulcer—

2051 traced cases

Proved ulcers

85 (4.14 per cent)

Proved and suspected ulcers

176 (8.63 per cent)

For Gastric Ulcer—

1382 traced cases

Proved ulcers

32 (2.32 per cent)

Proved and suspected ulcers

62 (4.48 per cent)

The first fact that emerges is that no patient with *carcinoma* developed a secondary ulcer, whatever operation he had undergone. There were 644 such cases, of whom 436 were traced. The possible explanations are (1) That carcinoma patients are immune from secondary ulceration, (2) That these patients do not live long enough for secondary ulcers to develop—this is only true in part, as secondary ulcers may appear very quickly, (3) Symptoms of secondary ulcer are likely to be masked by those of carcinoma. It is probable that secondary ulceration is a rare event after operation for carcinoma owing to the low acidity and probably also to the diminished peptic activity of the gastric juice.

Of cases of *duodenal ulcer* operated on we find that the incidence of secondary ulcer is just over 4 per cent, taking the cases successfully followed up and the number of secondary ulcers found at subsequent operation. If to these cases we add those where the presence of an ulcer was diagnosed on clinical or radiological evidence only, the incidence rises to over 8 per cent. This is much higher than the 2.8 per cent arrived at by Luff² in his investigation into the after-history of gastro-enterostomy, but that figure is clearly an underestimate even of the cases proved by operation, in the list of operations required for 'intercurrent' lesions after gastro-enterostomy for duodenal ulcer, no fewer than four are almost certainly cases of secondary ulcer—indeed, one patient had a gastrocolic fistula, and a similar criticism applies to gastro-enterostomy for pyloric ulcer and for perforated duodenal and pyloric ulcers.

In arriving at a true estimate of the incidence of secondary ulcer we are faced with the difficulty that there is a large number of untraced cases, more than one-third of the total, and it is impossible to know whether the proportion is the same for these untraced cases or not. From experience I am of opinion that it is less, as I believe that most patients return to the same surgeon when symptoms recur. We are also in a difficulty with those cases where the diagnosis rests on symptoms. In some of these the diagnosis is undoubtedly wrong, but in what proportion this is so it is impossible to say.

Taking all things into consideration, I think it is a fair estimate to say that secondary ulcer occurs in about 6 per cent of patients after posterior gastrojejunostomy for duodenal ulcer.

The figures for posterior gastrojejunostomy with entero-anastomosis are too small for any conclusions to be drawn from them, but as they stand they do not support the view that entero-anastomosis adds to the risk.

Anterior gastrojejunostomy seems to carry definitely a higher risk, and anterior

* The nomenclature of the operations in *Table I* includes the various modes by which they may be carried out and should be interpreted as follows: *Anterior Polya* = partial gastrectomy with antecolic end-to-side anastomosis. *Posterior Polya* = partial gastrectomy with retrocolic end-to-side anastomosis. *Billroth No. I* = partial gastrectomy with gastroduodenal end-to-end anastomosis. *Old type Billroth No. II* = partial gastrectomy with closure of the cut end of the stomach and gastrojejunostomy.

gastro-enterostomy with entero-anastomosis is especially harmful, although the figures again are very small in the latter condition. A study of the published cases many years ago convinced me that the combination of anterior gastro-jejunostomy with entero-anastomosis was a very deadly one, and these figures, small as they are, confirm me in my impression that this operation should not be used in ulcer cases. The small series of cases of gastroduodenostomy merely shows that this operation is not immune from secondary ulcer.

For the rest, it would appear that methods of gastric resection by the Polya methods are very much less liable to secondary ulceration than gastro-enterostomy. The numbers, however, are small, and it may be that a more general adoption of these operations would result in a higher incidence. Certainly further ulceration may and not infrequently does follow Polya gastrectomy for secondary ulcer.

It is difficult to estimate the risks of partial gastrectomy by the Billroth I method, as we have no cases proved by operation, and eight diagnosed on symptoms in a very small series. If the diagnosis is correct in any larger proportion of these, then the incidence is high.

Cases of *gastric ulcer* treated by posterior gastrojejunostomy in the present series developed proved secondary ulcers in 5.32 per cent, and the combined percentage of proved and suspected ulcers is 10.45 per cent. Both these percentages are definitely higher than the corresponding figures for duodenal ulcer patients.

This is a very unexpected finding, and one which is quite contrary to my personal experience. I believe that the explanation lies in the custom of including ulcers of the pyloric region among the gastric ulcers. I suspect that in a very large proportion of these cases the original lesion was a pyloric ulcer, i.e., one of the same nature as a duodenal ulcer, and which would be classified as duodenal by many surgeons. This view is supported by the fact that if we take the percentage incidence of all the post-duodenal cases, including all types of operation, we find that in 2051 traced cases there were 83 proved and 91 suspected secondary ulcers, viz., 4.05 per cent and 8.5 per cent—almost exactly the same as that for posterior gastro-enterostomy. For the post-gastric ulcer cases the corresponding figures are, total 1382, proved ulcers 33, suspected ulcer 30 giving 2.38 per cent and 4.55 per cent. Thus the percentage incidence over all operations is only about half that after posterior gastrojejunostomy for gastric ulcer.

Where other operations are concerned, such as gastrojejunostomy plus excision of ulcer, gastric resections, etc., the original ulcer was obviously of the true lesser-curve or gastric type, and the incidence of secondary ulceration is therefore small.

Other suggestive facts are found when a similar problem arises in connection with cases stated to have followed operation for gastric ulcer in the analysis of the returns of individual instances of secondary ulcer in *Tables II-VI*. It is convenient to deal with these facts here as the problem is the same. In 107 cases the original ulcer was said to be gastric, but of those 14 were definitely called pyloric and in 13 the operation was done at the time of an acute perforating ulcer, which must obviously have been situated at the pylorus to call for such an operation. In two more the operation was done some years after an acute perforation. This makes a total of 29 out of 107 where the ulcer was almost certainly pyloric, and probably others were of the same kind.

It seems probable, therefore, that in a considerable proportion of cases where the original ulcer was labelled gastric it was really pyloric or duodenal.

It is to be noted that a large proportion of gastric resection operations were for gastric ulcer and that the incidence of proved secondary ulcer is very small

In 622 resection operations (including local excision and gastrojejunostomy) for gastric ulcer there were 4 proved examples of secondary ulceration and 4 suspected, viz., 0.65 per cent proved and 1.30 per cent combined. In 146 for duodenal ulcer there were 1 proved case and 8 suspected. This gives 0.68 per cent proved cases and 7.5 per cent combined. The notable feature is the high incidence of suspected cases after operation for duodenal ulcer. The highest incidence after resection operations seems to be after operations of the Billroth I type.

Turning now to the analysis of actual cases of secondary ulcer, including cases of lesser-curve ulcer developing after operation for ulcer, we find 458 cases, after rejecting several where it seemed probable that the lesion was not a secondary ulcer, but an unhealed or recurrent gastric lesion. 107 of these followed operation for gastric ulcer, and I have already dealt with the question as to a possible fallacy in this figure. The men outnumber the women by about $3\frac{1}{2}$ to 1, and the condition is met with chiefly between the ages of 30 and 60, the peak incidence being the decade 40-50 (*Table II*).

Table II—AGE AND SEX INCIDENCE IN GASTRIC ULCER

		Males	Females
Under 20 years		0	0
20-30 years		4	3
30-40 years		25	2
Males	82	35	10
Females	24	14	6
Not stated	1	4	3
		0	0
	107	82	24
		1	
Not stated			

In comparison with the above, there were 351 cases where the primary operation was for duodenal ulcer. The chief difference between the two is the enormous disparity in the sex incidence in the latter, men outnumbering women by $21\frac{1}{2}$ to 1. There is not much difference in the age except that the proportion of cases under 30 is somewhat greater (*Table III*).

Table III—AGE AND SEX INCIDENCE IN DUODENAL ULCER

		Males	Females
Under 20 years		1	1
20-30 years		45	1
30-40 years		91	2
40-50 years		117	9
Males	328	59	1
Females	16	12	0
Not stated	7	3	0
	351	328	14
		14	
Add 9 cases, age not stated		9	
Stated		351	

The type of the original operation (*Tables IV, V*) was simple posterior gastrojejunostomy in a very large majority of cases, and the chief difference to be noted

between the post-gastric and the post-duodenal series is that the original operation was a partial gastrectomy of some kind in 8 out of 107 in the former and in only 1 out of 351 in the latter. This is of course explained by more common use of these procedures in cases of gastric ulcer. In the same way we account for the primary operation of gastrojejunostomy in combination with local excision or cautery of an ulcer. Entero-anastomosis figured in about 4½ per cent and pyloric occlusion in about 4 per cent.

Table IV—TYPE OF ORIGINAL OPERATION IN CASES OF GASTROJUNAL ULCER FOLLOWING OPERATION FOR GASTRIC ULCER

Posterior gastrojejunostomy	77
Posterior gastrojejunostomy with excision of ulcer	7
Posterior gastrojejunostomy with cautery of ulcer	4
Posterior gastrojejunostomy with pyloric occlusion	2
Posterior gastrojejunostomy with entero-anastomosis	3
Anterior gastrojejunostomy	6
Posterior Polya gastrectomy	2
Anterior Polya gastrectomy	2
Billroth I	1
Billroth II	1
Not stated	2
	<hr/>
	107

Table V—TYPE OF ORIGINAL OPERATION IN CASES OF GASTROJUNAL ULCER FOLLOWING OPERATION FOR DUODENAL ULCER

Posterior gastrojejunostomy	315
Posterior gastrojejunostomy with pyloric occlusion	10
Posterior gastrojejunostomy with entero-anastomosis	4
As above and pyloric exclusion	1
Anterior gastrojejunostomy	12
Anterior gastrojejunostomy with entero-anastomosis	8
Billroth II	1
	<hr/>
	351

It is frequently stated that gastrojejunostomy done at the time of an acute perforation is especially liable to secondary ulceration. From an examination of this series I find that in 351 gastro-enterostomies for duodenal ulcer the operation was done at the time of an acute perforation in 39—about 11 per cent. These findings support the view that secondary ulceration is specially apt to follow such a proceeding. In a further 13 instances the operation was done some time after suture of a perforation, so that perforation figures in 52 out of the 351 cases (*Table VI*).

Table VI—GASTROJUNAL ULCER FOLLOWING OPERATION FOR PERFORATION

Primary operation done at the time of perforation—	
<i>a</i> Perforated gastric ulcer	13
<i>b</i> Perforated duodenal ulcer	39
	<hr/>
	52

That is, 11 per cent of the total of 458 cases.

In 15 cases the primary operation was done at some period after an acute perforation. Therefore in 67 cases the original ulcer was a perforating one—14.6 per cent.

I have already stated that in the post-gastric ulcer series the operation was done for acute perforated ulcer on 13 occasions—just over 12 per cent—and on 2 occasions at some period subsequent to perforation. In the total of 458 secondary ulcers, therefore, the primary operation was done for acute perforation in 52—a

little over 11 per cent. On 15 occasions it was done at some period subsequent to perforation, giving a total of 67 cases in which perforation figured in the original lesion, or 14.6 per cent.

It is sometimes stated that when pyloric stenosis has supervened the liability to secondary ulceration is lessened, though anyone who has studied the early recorded cases would know that this is not so. As the information was not asked for in this inquiry, no conclusions can be drawn from the fact that it was mentioned as being present in 7 cases only.

SYMPTOMS (*Table VII*)

Pain* is the cardinal symptom of secondary ulcer. It is even more prominent than in ordinary gastric or duodenal ulcer, and is frequently more intense. It differs also in situation. It may be felt almost anywhere in the abdomen, either on the right side or the left, above or below the umbilicus, though in a very large proportion it is felt in the epigastric region, and when it is located to one side it is usually the left. In many patients the pain radiates over a wide area, being felt in several of the regions described. A curious feature is the relatively large number of cases in which the lower abdomen is affected.

A further point which emerges is that pain is seldom absent. In only 20 of the 458 cases was it definitely stated that pain was absent, and 14 of these were cases of colic fistula. I shall refer to this last point again.

The pain is usually severe in character, and does not exhibit the same precise features that it does in ordinary gastric or duodenal ulcer. Sometimes it is described as being continuous. The relationship to food is not so constant, but when present the onset is usually late, though this varies widely. Relief from ingestion of food is not very constant, but vomiting usually seems to give relief, so much so that in a fair number of cases it is said the patient induces vomiting in order to relieve the pain.

Vomiting occurred in half the cases. It relieved the pain, was sometimes self-induced, and varied much in frequency and intensity. The two conditions in which it was severe were when there was stenosis at the anastomosis and when a fistula into the colon existed. In the latter case the vomiting was often faecal.

Diarrhoea was noted in 55 cases, and many of these were patients with colic fistula. So far from being common, it is definitely stated to have been absent in 319 cases, and many of the reporters have gone out of their way to say that instead of suffering from diarrhoea, the patients have had extreme constipation. Where colic fistula existed, however, diarrhoea, often extreme, has been a constant symptom.

Wasting was more often met with than diarrhoea—in almost half the cases, but again it is a symptom which is especially in evidence where colic fistula exists.

Hæmorrhage occurred in 124 patients, and in 65 of these it was severe or repeated. It manifested itself both in the form of hæmatemesis and melæna, and figures as the actual cause of death in 9 cases. 2 of these are obvious

* In many cases the pain is described as widespread, extending from the upper to the lower abdomen. It is to be noted that the pain was stated to be in the left side in 93 cases and in the right in 24 only. It is also to be noted that of the 20 cases in which it was definitely stated that there was no pain, 14 were cases of fistula into the colon.

Table VII—SYMPTOMS AND SIGNS IN GASTROJEJUNAL ULCERATION FOLLOWING OPERATION FOR DUODENAL AND GASTRIC ULCER

<i>Pain—</i>	
Epigastric	245
Right epigastric	11
Left epigastric	44
Right hypochondrium	9
Left hypochondrium	20
Umbilical	23
Left flank	4
Lower abdomen	41
Left iliac fossa	25
Right iliac fossa	4
In the back	13
None	20
Not stated	14
<i>Type of Pain—</i>	
Continuous	13
Not related to food	69
Onset within half-hour after food	50
Onset $\frac{1}{2}$ –1 hour after food	50
Onset 1–2 hours after food	80
Onset more than 2 hours after food	67
Pain relieved by food	74
Not relieved	4
Night pain	46
Type of pain not specified	64
<i>Vomiting</i> (based on 411 cases, acute perforations excluded)—	
Present	229
Absent	157
Not stated	25
<i>Diarrhœa—</i>	
Present	55
Absent	319
Not stated	37
<i>Hæmorrhage—</i>	
Slight	59
Severe	65
Absent	265
Not stated	22
<i>Wasting—</i>	
Present	166
Absent	198
Not stated	47
<i>Tenderness—</i>	
Epigastric	145
Right epigastric	12
Left epigastric	45
Right hypochondrium	8
Left hypochondrium	9
Umbilical	12
Left umbilical	31
Right umbilical	6
Left iliac fossa	4
None	68
Not stated	118

Palpable lump noted in 22 cases only, and in only 7 of these had the original operation been anterior gastrojejunostomy

post-operative bleedings, but 7 occurred at intervals of three weeks to several years after operation, and are genuine examples of bleeding from a secondary ulcer. The only other symptom of any note is anæmia after severe or repeated hæmorrhage.

Tenderness on Palpation was present in more than half the cases. Generally it was elicited in the epigastrium, but it is noteworthy that it was found in almost every region of the abdomen. The left side of the abdomen was implicated much more commonly than the right side, and this preference for the left is more definite as regards tenderness than pain. Speaking generally, both pain and tenderness seem to be met over a much wider distribution than in primary duodenal or gastric ulcer.

It is somewhat surprising to find that a palpable lump was found on 22 occasions only, and that of these the primary operation was of the anterior type only in 7. This is very different from the reports of cases years ago, when a palpable tender lump under the left rectus muscle was commonly described and figured as an important point in the diagnosis. It is of course a fact that in the early days anterior gastrojejunostomy was much the commoner operation, and perforation into the abdominal wall tends to happen in secondary ulceration after this operation.

In this series there were only 28 cases of pre-colic operations, including 2 examples of anterior Polya gastrectomy, and of these 6 had acute perforating ulcers. There remain, therefore, 22 cases, in 7 of which a palpable lump was noted, roughly one-third. This confirms the old observation that a palpable mass is common after an anterior operation and rare after a posterior one, which is only what is to be expected when the deep situation of the posterior operation is remembered.

RADIOLOGICAL FINDINGS

Radiological investigation was carried out in 279 cases, and in these the findings were positive in 184, doubtful in 29, and negative in 66 (*Table VIII*).

Table VIII—RADIOLOGICAL INVESTIGATIONS IN 407 CASES OF GASTROJEJUNAL ULCER
(ACUTE PERFORATIONS EXCLUDED)

No examination in	128
Negative findings	66
Doubtful findings	29
Positive findings	184
	—
	407

Where details were given the positive diagnoses were made on the following appearances, or combinations of them —

Delay in emptying the stomach	20
Tenderness at the stoma	19
Residue at the stoma	25
Deformity at the stoma	17
Stenosis of the stoma	13
Crater	25
Gastrocolic fistula	25
Deformity in the jejunum	4
Ulcer of the stomach	7

In one case a Murphy's button was seen in the stoma.

ETIOLOGY OF SECONDARY ULCER (*Tables IX-XIII*)

We are no nearer solving the cause of secondary ulcer than we were twenty-five years ago, but our ideas are modified in some respects. At that time it was supposed that errors in technique were of great importance, and numerous suggestions were put forward, such as the use of a continuous unabsorbable suture, the pressure of clamps, the production of a hæmatoma in suturing, and in a general way sepsis of the anastomosis from lack of care in the suturing. Paterson thought that errors in technique probably were responsible for his gastrojejunal form of ulceration, but not for the true jejunal ulcers. An argument in favour of these views of the influence of technique was that the ulcers generally came within the first two years after the operation.

In our series we find that the onset of symptoms was within two years in 284 out of 458 cases—that is, just over 62 per cent (*Table IX*). This does not seem to me to favour the view that technique is a large factor in the production of secondary ulcer, as in such a large proportion the onset is so delayed that it is difficult to believe that technique can have had any influence. The shortest period was nine days in a patient who had a subacute perforation, and in 47 patients the interval which elapsed was less than one month. This suggests that in some cases at least technique has been at fault, but when we try to find any particular error there is none which can be found in any very large number of cases.

Table IX—PERIOD AFTER ORIGINAL OPERATION FOR DUODENAL OR GASTRIC ULCER AT WHICH SYMPTOMS OF GASTROJEJUNAL ULCER BEGAN (458 CASES)

Less than one month	47	5-6 years	15
1-3 months	40	6-7 years	10
3-6 months	61	7-8 years	8
6-12 months	70	8-9 years	5
1-2 years	66	9-10 years	9
2-3 years	36	Over 10 years	15
3-4 years	30	Not stated	29
4-5 years	17		

Thus in 109 cases more than three years elapsed before the onset of symptoms, roughly 25 per cent of the cases where the information was available. The shortest period was 9 days.

Table X—INTERVAL BETWEEN ORIGINAL OPERATION FOR DUODENAL OR GASTRIC ULCER AND FIRST OPERATION FOR GASTROJEJUNAL ULCER (458 CASES)

Less than one month	5	5-6 years	28
1-3 years	6	6-7 years	27
3-6 months	12	7-8 years	15
6-12 months	39	8-9 years	19
1-2 years	90	9-10 years	13
2-3 years	63	Over 10 years	38
3-4 years	36	Not stated	26
4-5 years	41		

Unabsorbable suture material, which I myself at one time blamed, was found on 28 occasions in the 279 cases where the information was available—about 10 per cent—and it is interesting to note that in one of these catgut was still unabsorbed at the end of two and a half years. On 3 occasions a metallic foreign body was present, one being a Murphy's button. The mere presence of unabsorbed suture material is not proof that it was the cause of the ulceration. If present, it would

naturally be exposed during the process of ulceration. If this were really a frequent cause of secondary ulcer, one would expect that the use of absorbable sutures would materially lessen the risk, but we find that in a series of 272 where the technique was given, in 214 catgut alone was employed. The use of an absorbable suture is therefore no safeguard against secondary ulcer.

There are only 9 cases where clamps were dispensed with (*Table XI*), but in view of the very general use of clamps I do not think too much stress can be laid on this fact. I have myself for some years avoided the use of a clamp on the jejunum because I found that if it was applied sufficiently tightly to control the bleeding it was apt to bruise the bowel.

Freak happenings, like the presence of a Murphy's button, need not be considered, as few surgeons would employ such a technique nowadays.

It seems difficult, then, to point to any particular error in technique which can be blamed as a common cause of secondary ulcer, though probably it is a secondary factor in some cases.

Table XI—TECHNIQUE EMPLOYED IN ORIGINAL OPERATION FOR GASTRIC OR DUODENAL ULCER

Clamps and continuous catgut suture both rows	210
Clamps and continuous catgut inner layer, with continuous silk or thread outer layer	45
Clamps and continuous silk or thread both layers	7
Clamps, interrupted catgut and thread	1
No clamps and continuous catgut both layers	4
No clamps and continuous catgut inner layer with continuous silk or thread outer layer	5
Murphy's button	1
Not known	185
	<hr/> 458

Table XII—SUTURE MATERIAL OF ORIGINAL OPERATION AT TIME OF SECONDARY OPERATION

Absent	246
Present (in one case catgut after 25 years)	28
Piece of needle	1
Piece of steel	1
Murphy's button	1
Not stated	181
	<hr/> 458

That the production of these ulcers depends in some way on the action of the gastric juice is agreed on all hands. Paterson made a very able special plea for hyperacidity as the chief cause, but this does not seem to me to be in accordance with the facts, even those on which he based his opinion. In our series hyperacidity was present in only about half the cases when investigations were made, and in 8 cases there was actual achlorhydria (*Table XIII*).

Although we may agree that hyperacidity is a common accompaniment of secondary ulcer, it is difficult to believe, in view of these findings, that it is the actual cause. It may indeed be the result. Even if we accept it as the cause, we are not much better off, because we are still in the dark as to the cause of the hyperchlorhydria.

That the gastric juice is essential in the production of these ulcers is, however, certain. The experimental work of Matthews and Dragstedt,³ who exposed the mucosa of the small intestine to the action of unneutralized gastric juice, shows

conclusively that in such conditions chronic peptic ulcer follows inevitably. Their work also shows the importance of concentration of the gastric juice and the influence of stasis in increasing that concentration, and confirms the value of reflux of bile and pancreatic juice in neutralizing the gastric juice, an observation made many years ago by Boldyreff and confirmed by Paterson, who believes this to be the chief physiological action of gastrojejunostomy. Owing to the ease of estimating the acidity of the gastric juice has attracted much more attention than the peptic activity, but it is well to remember that the latter is inactivated by the action of bile and pancreatic juice, just as the former is neutralized.

Table XIII—CONDITION OF GASTRIC JUICE IN GASTROJEJUNAL ULCER

The acidity of the stomach contents after a test-meal was estimated in 138 cases as follows —

Hyperchlorhydria	64
High normal acidity	12
Medium normal acidity	27
Low normal acidity	27
Achlorhydria	8

The normal range of free HCl is taken as 20–40

Similar investigations at the time of the primary operation in 66 cases gave the following results —

Hyperchlorhydria	32
Normal acidity	31
Achlorhydria	3

It can be agreed, therefore, that gastric juice is essential for the production of chronic secondary ulcer, and that its noxious influence is greatly increased by any stagnation or interference with its exit from the stomach or anything which prevents the reflux of bile and pancreatic juice. For these reasons the gastro-enterostomy should be well placed and of sufficiently large size. If it is too much towards the cardiac end, it will probably not empty the stomach very efficiently. Caution must, however, be used in deciding that the gastro-enterostomy opening has been too small originally. The presence of secondary ulceration on the anastomosis leads to considerable contraction of the stoma, so that an opening which was originally quite large becomes small and inadequate.

Operations en-Y and with entero-anastomosis should not be employed in the treatment of ulcer cases, either as primary or secondary operations, since they expose the jejunal mucosa to unneutralized gastric juice. The true etiology is unlikely to be known until we have solved that of duodenal and gastric ulcer. Whatever that cause, it is probably still active in the case of secondary ulcer.

MORBID ANATOMY

The information received was not sufficiently detailed to enable me to give anything but a broad general description of the lesions found.

Site of Gastrojejunal Ulcer (*Table XIV*)—The first point which emerges is the enormous preponderance of anastomotic ulcers—285, as against 99 jejunal ulcers. There was not enough detail to allow me to give the exact location of the ulcers on the anastomosis, but it is evident that sometimes the ulceration extends right round the anastomotic ring. When it is more limited it tends to exist at the

angles, and in my own experience I have found that the proximal angle of the anastomosis is a favourite site. I had an impression that jejunal ulcer was more likely to follow anterior gastrojejunostomy, and anastomotic ulcer the posterior operation, but that is not the case. True jejunal ulceration is found in the efferent loop in the great majority of cases. It may be situated within $\frac{1}{2}$ in. of the anastomotic ring or 1 to 2 in. or more away from it. The situation in the afferent loop

Table XIV—SITE OF GASTROJEJUNAL ULCER * (458 CASES)

Anastomotic	285
Jejunal, exact site not stated	6
Jejunal Afferent loop	14
Jejunal Efferent loop	68
Jejunal Mesenteric border	11
Gastric Near anastomosis	1
Gastric Extending from anastomosis	30
Jejunal Extending from anastomosis	14
Anastomotic and lesser curve ulcer	11
Anastomotic and separate jejunal ulcer	1
Independent ulcer lesser curve	5
Situation not stated	12

* This refers only to the ulcer operated on by the reporter of the case

is quite rare. An interesting group of cases is that in which a jejunal ulcer is found on the mesenteric border of the bowel, directly opposite the stoma of the gastro-enterostomy. This is usually quite independent of the anastomosis, and may produce obstruction by contraction drawing up a spur of bowel into the stoma. An ulcer was found in this situation on 11 occasions, so that it is a rare variety.

Complications (*Table XV*)—Perforation of a secondary ulcer may be acute into the general peritoneal cavity, or chronic. After an anterior gastro-enterostomy the secondary ulcer tends to become adherent to the anterior abdominal wall and to penetrate the rectus sheath, giving rise to a tender mass. In neglected cases the perforation may extend through the anterior abdominal wall and give rise to an external fistula.

Table XV—COMPLICATIONS OF GASTROJEJUNAL ULCER

Acute perforation	51
Fistula into colon	40
Perforation into abdominal wall	16
Perforation into mesocolon	21

After posterior operations there is a tendency to chronic perforation into the mesocolon, and the latter becomes thickened and contracted. In this way the colon becomes pulled down and held tightly against the thickened ulcer. In course of time a fistula may form between the two. This happened in 40 cases in the series. The fistula may be between the stomach and the colon—gastro-colic fistula, between the anastomosis and the colon—gastro-jejunocolic fistula, or between the jejunum and the colon—jejuno-colic fistula. After a fistulous opening has become well established, there is a tendency for the ulceration to heal, a fact to which I shall revert later.

In certain cases the ulceration spreads from the anastomosis to the stomach on the one hand or the jejunum on the other. Thus we find extension to the stomach in 30 cases and to the jejunum in 14. The extension from the

anastomosis may be over a long extent, and *Fig 291* shows a picture of a lesser-curve ulcer of the stomach connected to the anastomosis by a track of ulceration. It is said that all roads lead to Rome, and this shows the tendency of gastric ulceration to make for the lesser curve.

In 11 patients anastomotic ulcer and an independent lesser-curve ulcer existed together, but 8 of these followed operation for gastric ulcer and are probably examples of unhealed or recurrent ulcers. In the other 3 the gastric and anastomotic ulcers are both post-operative. These cases represent an intermediate stage on the way to the following type, where an independent ulcer formed on the lesser curve after gastrojejunostomy for duodenal ulcer. This has happened on 5 occasions in the present series. I have been aware of this possibility for a good



FIG 291 —Lesser curve ulcer of the stomach connected to the anastomosis by a track of ulceration

many years now. Though at first I thought it was an accidental occurrence, yet it influenced my views on the value of gastro-enterostomy as a treatment for lesser-curve ulcer. I have now seen a sufficient number of cases to convince me that the development of a lesser-curve ulcer after gastro-enterostomy for duodenal ulcer or pyloric stenosis is a phase of secondary ulceration. I would liken these ulcers to those on the mesenteric border of the jejunum. Usually they are situated directly opposite the stoma, though occasionally they are higher up. Further evidence of the formation of these ulcers after gastro-enterostomy is to be found from a study of Luff's paper, to which I have already referred. He reports 3 cases of lesser-curve ulcer after gastro-enterostomy in a series of 744 cases of operation for duodenal ulcer, and 2 cases in 454 similar operations for pyloric ulcer.

I have very little doubt that the alteration in the physiology of the stomach in some way leads to an increased liability to ulceration of the lesser curve.

TREATMENT OF SECONDARY ULCER

In spite of the fact that in many of these patients a secondary ulcer may exist for years, the majority are so miserable and the symptoms are so little relieved by medical treatment that resort to operative measures becomes imperative. Acute perforation of course demands immediate intervention, and repeated hæmorrhage is another indication for operation. In most cases, however, operation is demanded for the relief of the constant intolerable pain. The operations may be classified as follows —

1 **Local Operations** (*Table XVI*) — For the most part these are unsatisfactory, most of the patients being unrelieved and many requiring further operations. It is interesting to note that when the operation is confined to removal of a silk suture, no relief was obtained.

Table XVI — LOCAL ATTACK ON ULCER, WITH OR WITHOUT PLASTIC OPERATION ON THE STOMA

<i>Excision—</i>	
Good	5
Fair	1
Symptoms not relieved	5
Further operation	17
Late result not known	14
Died	3
Died later	2
	<hr/>
	47
<i>Excision of Affected Loop of Jejunum—</i>	
Good	1
Fair	0
Symptoms not relieved (died later of 'cancer')	1
Further operation	1
Late result not known	2
Died	1
	<hr/>
	6
<i>Transgastric Caутery of Ulcer—</i>	
Good	2
Further operation	2
Late result not known	2
Died	0
	<hr/>
	6
<i>Removal of Murphy's Button—</i>	
Good result	1
<i>Removal of Silk—</i>	
Not relieved	2
<i>Local Excision of Lesser-curve Ulcer—</i>	
Late result not known	1
Further operation	1

2 **Reconstitution of the Normal Alimentary Passage** (*Table XVII*) — Under this heading proceedings of widely different severity are included. In its simplest form it consists of the undoing of the gastro-enterostomy and the closure of the openings in the stomach and jejunum. In others it includes a resection of the affected loop of jejunum with end-to-end or lateral anastomosis. In one case the upper end of the jejunum was closed and the continuity of the alimentary canal re-established by anastomosing the lower end of the jejunum to the third part of the duodenum. This operation of restoration to the normal is a formidable

one and involves what is really the most difficult part of any operation for secondary ulcer—the freeing of the anastomosis from the mesocolon. It carries with it, therefore, a fairly high mortality. The results are disappointing. In only about 20 per cent was a known cure obtained, while 12 per cent required further operation, and one case died as the result of a perforation of a duodenal ulcer later. In 14 per cent the symptoms were unrelieved. The trouble is, of course, that the original duodenal ulcer tends to recur. The operation is only suitable for cases with a widely patent pylorus and duodenum, and should be reserved for those in whom no scar, or only a very slight one, is present in the duodenum.

Table XVII—RECONSTITUTION OF NORMAL ALIMENTARY PASSAGE

<i>Simple Undoing of Gastro-enterostomy and Re-constitution—</i>	
Good	18
Fair	7
Unrelieved	13
Further operation	11
Late result not known	30
Died	11
Died later of perforation	1
	<hr/>
<i>Excision of Gastrojejunostomy with Gastroduodenostomy or Pyloroplasty—</i>	91
Good	5
Fair	1
Unrelieved	3
Further operation	2
Late result not known	4
Died	0
	<hr/>
<i>Finney's Pyloroplasty—</i>	15
Not traced	1
<i>Gastroduodenostomy—</i>	
Recovered	1
Died	1
<i>Pyloric Occlusion—</i>	
Further operation necessary	1

Where an active duodenal ulcer still exists, or where the duodenum is stenosed, the operation must be combined with gastroduodenostomy or some form of pyloroplasty. This operation is spoken of highly by some, but the numbers in the present list are too small for a definite opinion to be formed. It is noteworthy, however, that one-third of the patients were unrelieved or required a further operation.

3 **New Gastro-enterostomy** (*Table XVIII*)—Under this heading are included cases where the old gastro-enterostomy has been resected and resutured, an extension of the first type of operation, and those where after resection and closure of the old gastro-enterostomy an entirely new one has been established.

Table XVIII—NEW GASTROJEJUNOSTOMY

<i>New Posterior Gastrojejunostomy—</i>	
Good	3
Fair	1
Unrelieved	1
Further operation	4
Late result not known	3
Died	6
	<hr/>
	18

<i>New Posterior Gastrojejunostomy with Entero-anastomosis—</i>	
Good	2
Fair	0
Unrelieved	3
Further operation	3
Late result not known	4
Died	2
Died later	4
	<hr/>
	18
<i>New Anterior Gastrojejunostomy—</i>	
Good	5
Fair	0
Unrelieved	0
Further operation	3
Late result not known	6
Died	0
Died later	1
	<hr/>
	15
<i>New Anterior Gastrojejunostomy with Entero-anastomosis—</i>	
Good	1
Fair	0
Unrelieved	0
Further operation	0
Late result not known	2
Died	1
	<hr/>
	4

Finally, in a few cases a new anastomosis has been done, leaving the old one intact. It will be noted that there was an operative mortality of 16 per cent and that quite a number of patients died later, especially in the series of new posterior gastrojejunostomy with entero-anastomosis. The percentage of good ultimate results is only 20.

4 **Entero-anastomosis** (*Table XIX*)—This was only rarely done and has nothing to recommend it in the small series. It offends against all our ideas on the causation of secondary ulcer.

Table XIX—ENTERO-ANASTOMOSIS

Good	2
Unrelieved	1
Further operation	2
Late result not known	3
Died	2
Died later of hæmorrhage	1
	<hr/>
	11

5 **Various Methods of Gastrectomy** (*Table XX*)—These operations offer by far the best chance of a cure—about 60 per cent, which is much higher than any other form of operative treatment. As against this, the operative mortality is very high, especially after the *posterior Polya operations*, when it approximates to 20 per cent. The *anterior Polya operation* shows a lower mortality—15.5 per cent—but the results are not quite so good as those from the posterior operation, 1 patient dying later and 3 requiring a further operation. I cannot help feeling that the mortality from these operations should be reduced. The difficult and dangerous part of the operation is that which is shared by all procedures which interfere with the anastomosis, i.e., the freeing of the anastomosis from the mesocolon and the resection of the anastomosis. When this has been accomplished the resulting gastrectomy is easy.

If recurrence takes place after a *partial gastrectomy* operation, any further operative treatment becomes most difficult and dangerous. In order to minimize the possibility of this, a gastrectomy for secondary ulcer should be made as high as possible.

Table XX—GASTRECTOMY BY VARIOUS METHODS

Posterior Polya Gastrectomy—

Good	49
Fair	4
Unrelieved	2
Further operation	3
Late result not known	7
Died	16

81

Posterior Polya with Entero-anastomosis—

Good	0
Fair	0
Unrelieved	2
Further operation	1
Late result not known	4
Died	0

7

Anterior Polya Gastrectomy—

Good	25
Fair	1
Unrelieved	0
Further operation	3
Late result not known	8
Died	7
Died later	1

45

Polya Gastrectomy (type not stated)—

Good	8
Fair	3
Unrelieved	1
Further operation	0
Late result not known	4
Died	4

20

Partial Gastrectomy (type not stated)—

Good	0
Fair	1
Unrelieved	0
Further operation	2
Late result not known	3
Died	2

8

Billroth No. I—

Good	1
Fair	0
Unrelieved	0
Further operation	1
Late result not known	3
Died	0

5

6 Devine's Operation (Table XXI)—Devine's operation of unilateral exclusion, which consists of a division of the stomach above the anastomosis, closure of the lower end, and anastomosis of the upper end to the side of the jejunum,

offers an easier alternative to partial gastrectomy. This was used in only 2 cases in the present series. In principle there seems to be no reason why it should not prove eminently successful.

Table XXI—DEVINE'S OPERATION

Good result	1
Not traced	1
	<hr/>
	2

7 Laparotomy (*Table XXII*)—One feature is the number of patients in whom an exploratory laparotomy was the first of the secondary operations. These operations usually ended in the breaking down of adhesions, and it is not surprising that the results were almost uniformly unsatisfactory, and that most of the patients underwent further operations subsequently.

Table XXII—LAPAROTOMY

Good	1
Fair	1
Unrelieved	3
Further operation	11
Died	1
	<hr/>
	17

8 Operations Impossible to Classify—These are summarized in *Table XXIII*.

Table XXIII—VARIOUS OPERATIONS IMPOSSIBLE TO CLASSIFY

Ligature of vessels—2 cases requiring further operation
 Jejunostomy—4 cases 3 died, 1 not traced
 Excision of duodenal ulcer for hæmorrhage—1 case requiring further operation

Nature of Operation not Stated—

Good	1
Unrelieved	3
Further operation	3
Died	2
	<hr/>
	9

In Addition, the Following Operations were Done—

Closure of perforated duodenal ulcer	2
Decapsulation of the kidney	1
Removal of cystic ovary	1
For appendix abscess	1
Cautery of gastric ulcer	2
Pylorotomy for lesser-curve ulcer	1

SUMMARY OF GENERAL RESULTS OF OPERATIVE TREATMENT IN SECONDARY ULCERATION

The study of *Table XXIV* is a truly saddening experience and one which reveals this complication as a really black spot in surgery. It shows that at the first attempt a reasonably good result was obtained in 31 per cent, but that 16 per cent required further operation, while almost as many died as the result of operation. In addition 12 patients died later as the result of the ulceration. Of the 76 patients who required further operation or operations I have tabulated the results to the present time. It will be seen that while about half of these have been restored to reasonable health, a further 18 died. From a combination of the two tables we

find that in 39 per cent there is a reasonably good result, while in more than 9 per cent the symptoms are known to be still present. Counting operative deaths and those who died from secondary ulcer though not as a result of operation, there were 104 deaths. This means that 22.7 per cent of patients suffering from secondary ulcer are known to have died as the result. The complication of secondary ulceration is therefore a truly disastrous one.

Table XXIV—RESULTS OF OPERATIVE TREATMENT OF SECONDARY ULCER

1 Results of First Secondary Operation—	
Good	120
Fair	23
Still symptoms	34
Required further operation	76
Not known	118
Died from operation	73
Died later	12
	<hr/>
	456
2 Results in the 76 Cases After Further Operations—	
Good	30
Fair	6
Still symptoms	10
Not traced	12
Died	18
	<hr/>
	76
3 Combination of 1 and 2 in the General Results—	
Good	150
Fair	29
Symptoms still present	44
Later results unknown	130
Died as result of operation	91
Died of the disease	12
	<hr/>
	456

In addition 1 patient died of acute perforation before operation could be done, and 1 case was discovered at a post-mortem for an intercurrent disease.

ACUTE PERFORATING SECONDARY ULCER (Table XXV)

Of the 458 cases there were 48 in which the first secondary ulcer was met with as an acute perforation into the general peritoneal cavity. In 42 of these the primary operation had been posterior. Since of the total of 458 cases the primary operation was posterior in 430, this gives a percentage of 9.7. In the remaining 6 the primary operation was of the anterior type. As there were only 28 in which this was the case, the percentage of acute perforations is about 21. From this it would appear that secondary ulcers after an anterior primary operation are more liable to be of the acute perforating type than those following a posterior operation. In addition to the cases which first presented themselves as acute perforations, there were 12 patients in whom an acute perforation occurred at some period after an operation for secondary ulcer, giving a total of 60 cases to be considered.

In 48 the operation consisted of suture of the perforation, with or without drainage of the peritoneum. Of these, 11 died—almost 25 per cent—and only a very small number recovered to be free from symptoms. In 15 further operation proved necessary later.

In 4 patients drainage of the peritoneum only was the operative treatment adopted. As was to be expected, the mortality was 100 per cent.

In a small number of cases entero-anastomosis was done at the time of the suture of the perforation. These were not followed up properly, but on general principles the operation is a bad one.

In 2 patients restoration to the normal alimentary passage was done at the time of the perforation. One died and one had recurrence of symptoms.

The operation of choice is undoubtedly simple suture of the perforation, with or without drainage of the peritoneum according to circumstances. Many will require further operative treatment, but a trial should be made of prolonged dietetic treatment after this simple operation.

Table XXV—OPERATIONS FOR ACUTE PERFORATIONS

<i>Suture with or without Drainage—</i>	
Good	5
Fair	3
Symptoms recurred	6
Further operation	15
Not traced	8
Died	11
	<hr/>
<i>Drainage Only—</i>	48
Died	4
<i>Closure of Perforation and Entero-anastomosis—</i>	
Fair result	1
Further operation	1
Later result not known	3
	<hr/>
	5
<i>Closure of Perforation and Reconstitution of Normal Passage—</i>	
Symptoms recurred	1
Died	1
	<hr/>
	2
<i>Died without Operation</i>	1

GASTROCOLIC FISTULA

Fistula into the colon was present when the patient sought treatment in 40 cases—34 in the post-duodenal series and 6 in the post-gastric. In all of these the primary operation was of the posterior type, but it is noteworthy that in addition to these there were 8 examples of colic fistula following operation for secondary ulcer, and in 2 of these the condition followed an anterior Polya gastrectomy. In both these the fistula was of the jejunocolic type and obviously formed at the place where the jejunum crossed the colon. These two examples are the only cases I have ever known to develop a colonic fistula after an antecolic operation, and it would seem to be due to the peculiarly intimate relationship of the jejunum to the colon after an anterior Polya operation. Cases following gastrojejunostomy have invariably been after the retrocolic operation.

Symptoms (*Table XXVI*)—The symptoms of gastrocolic fistula differ in several important respects from those of ordinary secondary ulcer.

While *pain* is the cardinal symptom of ordinary secondary ulcer it was completely absent in 15 of the 40 cases, and slight only in 8. Thus in 23, or more than

half the cases, pain was a negligible symptom. In only 13 was pain noted as a symptom of any prominence, and in only 4 of these was it said to be severe. No note was made in 4. In one patient the observer stated that pain was a prominent symptom for four years from the time of the original operation, but that suddenly with the onset of fæcal vomiting, diarrhœa, and wasting the pain ceased.

I believe the explanation of this absence of pain is the healing of the active ulceration after the establishment of a fistula into the colon. I have noted at operation on such a case that the very wide communication between the anastomosis and the colon was completely healed and covered with mucosa.

The trilogy of symptoms on which the diagnosis rests is *vomiting, diarrhœa, and wasting*, all three or some of which are invariably present. When the vomiting is fæcal in type the diagnosis is easy, and even when this is not the case the patient frequently complains of foul eructations.

Table XXVI—SYMPTOMS IN GASTROCOLIC FISTULA

<i>Pain—</i>	
Absent	15
Slight	8
Present	9
Severe	4
Not noted	4
	<hr/>
	40
<i>Vomiting—</i>	
Fæcal	11
Severe	10
Present	9
Absent	5
Not stated	5
	<hr/>
	40
<i>Diarrhœa—</i>	
Severe	18
Present	16
Absent	4
Not stated	2
	<hr/>
	40
<i>Wasting—</i>	
Severe	11
Present	19
None	6
Not stated	4
	<hr/>
	40

Treatment (Table XXVII)—Operative treatment is urgently necessary in this condition. Even though the patient may be free from pain, his condition is miserable and clamorous for relief.

It will be seen that the operative procedures are similar to those adopted in ordinary secondary ulcer, but there is the added complication of separation and closure of the hole in the colon. In some cases this has called for a resection of the colon in addition to the manipulations necessary for the treatment of the secondary ulcer. In such a case if the treatment adopted is one of partial gastrectomy, it may involve resection and end-to-end union of the jejunum, resection of the colon, resection of the stomach and anastomosis to the intestine. This is a

truly formidable operation, and if it is not really possible to cobble up the hole in the colon, it would be better to treat the colon by means of a double-ended colostomy and so save a considerable and risky part of the operation

Table XXVII—SECONDARY OPERATIONS FOR GASTROCOLIC FISTULA

<i>Separation of Viscera and Closure of Openings, with or without Formal Excision of Ulcer—</i>	
Good	1
Fair	2
Unrelieved	1
Further operation	3
Not traced	4
Died	5
Died later	1
	—
	17
<i>As above, with Entero-anastomosis—</i>	
Died 4 years later after much suffering	1
<i>Resection of Colon and New Gastrojejunostomy—</i>	
Unrelieved	1
Further operation	3
Died	1
	—
	5
<i>Separation of Fistula Reconstitution of Normal Alimentary Passage—</i>	
Good	6
Unrelieved	1
Further operation	1
Not traced	1
Died	4
	—
	13
<i>Posterior Polya Gastrectomy with Repan or Resection of Colon—</i>	
Good	4
Not traced	2
Died	2
	—
	8
<i>Anterior Polya Gastrectomy and Repair of Colon—</i>	
Requiring further operation	1
Not traced	1

In addition, 2 cases were operated on for colic fistula after posterior Polya gastrectomy. One of them was of the posterior Polya type en-Y. The anastomosis was excised and anterior Polya performed. Patient had a perforated jejunal ulcer later. In the other the edges of the anastomosis were refreshed and resutured. Later result unknown.

In these complicated cases milder operations, such as restoration of the normal alimentary passage, may well be adopted, and in the small series where this was done, almost half were cured. Whatever operative treatment is required it remains one of the most severe and trying operations in surgery.

REFERENCES

- ¹ PATERSON, H. J. *Proc Roy Soc Med*, 1908-9, 11, 238
- ² LUFF, *Brit Med Jour*, 1929, 11, 1074
- ³ MATTHEWS and DRAGSTEDT, *Surg Gynecol and Obst*, 1933, 14, 265

CHRONIC INTERSTITIAL MASTITIS

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THIS paper is entitled 'chronic interstitial mastitis' because that is the name by which the condition of the breast which it denotes is most familiarly known in this country. It is otherwise known as 'chronic cystic mastitis', 'cystic disease of the breast', 'Schimmelbusch's disease', 'Brodie's disease', 'chronic lobular mastitis', and a host of other names. A great deal of recent work on this condition, however, goes to prove that it is not of inflammatory origin and is only secondarily interstitial. The name 'chronic interstitial mastitis' should, therefore, now be abolished and a more appropriate name adopted, such as 'mastopathia' which was first suggested by Aschoff¹ and has recently been revived by Whitehouse².

I am attempting to give a description of the disease and to put forward a theory of its causation which is based on the work of those who have devoted many years of research to this subject. This theory has been tested, first by the findings in an examination (which Mr R W C Murray, the Surgical Registrar, and myself have made) of the breasts of 500 consecutive female admissions to the Queen's Hospital, Birmingham, secondly, by a series of microscopical examinations of the breasts of female post-mortem cases irrespective of the cause of their death, and, lastly, by the results in the treatment of a series of cases of painful nodular breasts. In many diseases of obscure origin the trend of scientific opinion is carrying us away from the long-established idea of a focal infective causation to one which blames an imbalance of the internal secretions. The fault, it seems, of chronic illness lies not in our teeth, tonsils, or appendices, but in our glands, deficiencies in whose secretion gives rise to faults in the physiology of different organs of the body, and it is in this theory that the key to this difficult and contentious problem of breast pathology is, in my opinion, to be found.

I do not wish in this short paper to give a history of the work done on this disease, but the remarks of two early authors, Creighton and Reclus, are of importance, in that their conclusions are the same essentially as those arrived at after careful study by Cheatle and Keynes to-day.

Creighton³ in 1878 first propounded the theory that the retained secretions in the breast were the chief factors in causation of tumours of the breast. This may be called the 'stagnation theory' and is upheld by Keynes¹ in this country and by Bertels⁵ and Lukowsky⁶ on the Continent. Reclus,⁷ writing of the disease in 1883, called it 'intra-acinous cystic epithelioma'. He came to the conclusion that the cysts resulted from epithelial proliferation in carcinoma with degeneration of those cells and cyst formation. Cheatle's idea of cyst production, it will be seen, is a modification and elaboration of this. Cheatle⁸ himself has introduced a completely new line of thought by dividing the condition into two separate varieties which he calls 'mazoplasia' and 'cystiphorous desquamative hyperplasia'.

respectively, and the following is an account of these conditions based on his descriptions

Mazoplasia is an essentially physiological process and is an epithelial hyperplasia and desquamation of the ducts and acini of the breast, mainly affecting the terminal and smaller ones. The periductal fibrous tissue increases and there is a lymphocytic infiltration of the interstitial tissues. Clinically there is said to be a diffuse aching pain over the whole of the breasts, often worse at menstruation and after excessive use of the arms. Women who suffer from this condition generally show evidence of ovarian hypofunction, their menstrual periods being of short duration and of scanty flow, and are, moreover, generally in poor health. In women with little or no subcutaneous fat, careful palpation can detect a fine state of nodularity of the anterior surface of the gland, which feels more solid than usual. This solidity may be uniform or more pronounced in one area than another. Transillumination of the breast is said to show a certain degree of opacity over the whole gland, but a trial of this method in our hands and in the hands of others whom I have consulted has proved of little or no value. This condition, according to Cheatle, is a definite separate clinical entity, and has no connection with the other variety—cystiphorous desquamative hyperplasia. Here the predominant feature is the formation of cysts, and it is said by Cheatle to have a different pathology.

Histologically, epithelial changes are seen either in the ducts alone or more commonly in the ducts and acini. In the ducts the cells become elongated, feathery, and form colostrum-like corpuscles in certain areas. Expansion of the ducts into cysts occurs. In the acini desquamation of epithelium occurs, the cells first of all becoming elongated and characteristically pale. These pale cells crowd in on the lumen, eventually disintegrate, and form small cysts, which, as the condition advances, coalesce with neighbouring cysts to form larger and larger ones. Clinically this type presents itself in three ways: first, as a single large cyst with the rest of the breast apparently normal; secondly, as a localized cystic change confined to one sector of the breast; and, lastly, as multiple small cysts scattered throughout the breasts. These, then, are the two varieties—mazoplasia and cystiphorous desquamative hyperplasia—entirely separated from each other by Cheatle.

In this paper, however, the theory is offered that mazoplasia, which is said to be a very common condition, may eventually in certain instances lead on to the cystic form of breast epithelial desquamation, and that all these breast changes are due to a faulty physiology. In other words, that all forms of non-malignant nodularity, whether cystic or not, are due to a breakdown in the hyperplasia involution cycle occurring in the breast with each intermenstrual period and leading to a physio-pathological process of excessive epithelialization and cyst formation. The thyroid is an organ which, like the breast, undergoes cyclical changes from one menstrual period to another, and it may also, owing to a faulty physiology, undergo parenchymatous changes, leading to the formation of a goitre where there is an increased formation of acini. This stage may progress by involution carried too far or not far enough to the formation of nodular areas which may show increased acinar formation or increased colloid distension. So may the breast, then, form a parenchymatous nodularity which may progress to a cystic state. The cysts which are formed may show the characters of a hypo-involution with increased epithelial lining, or of a hyper-involution where there is one single layer of flattened

epithelium In other words, the breast may pass from the nodularity of mazoplasia to the cystic form of breast hypo-involution, and give rise, as in the thyroid, to a generalized or local cystic change or to one large cyst

Put briefly, the theory we have to test is that mazoplasia is a common condition due to a faulty physiology which may eventually become so disordered that a further cystic change results

It would be opportune at this point to review the normal physiology of the human breast so that it may be seen how on this thesis a perversion of that physiology may give rise to a nodularity of the breast which is at first not cystic but may eventually become so

It has been shown conclusively that the breast responds harmonically to ovarian activity (Parkes and Bellerby,⁹ Loeb and Hesselberg¹⁰) At puberty a definite development occurs, and during each menstrual cycle the mammary glands pass through changes characterized by hyperæmia, increase in inter-alveolar stroma, and formation of alveoli This activity stops and the epithelium involutes in harmony with the growth of the corpus luteum Rosenberg,¹¹ Polano,¹² and Dieckmann¹³ have demonstrated these changes in the human breast, and Whitehouse² has recently pointed out that they bear a close analogy to the menstrual changes seen in the endometrium The factor governing the hyperplastic factor of the cycle in the breast epithelial changes is the hormone produced by the Graafian follicles Excessive epithelialization may, therefore, be due immediately either to excessive luteal activity or defective function of the rest of the ovary As, however, there is an harmonic balance between the endocrine glands, it is possible that the remote cause may lie in the anterior pituitary, which exercises a controlling effect upon ovarian activity In this connection it is interesting to note in passing that A P Thomson¹⁴ has shown the effect of irradiation of the pituitary in relieving menstrual headaches

Let us now trace the changes which may arise in the breast as a result of ovarian hypo-function During the hyperplastic period of the menstrual cycle, epithelial proliferation occurs in the acini, and new alveoli are formed In the normal breast this activity dies down and complete involution occurs Where there is an ovarian hypo-function this involution is not complete, and a new menstrual cycle begins before the last one has, so to speak, cleared up A further formation of epithelium occurs, the acini become packed and distended with cells, the delicate balance of secretion and absorption is upset, and fluid begins to collect in the terminal ducts and acini, giving rise to what may be called a 'precystic' distension (*Figs 292, 293*) This condition may remain in this stage and give rise to nothing more than a persistence of the lumpiness that is more or less normal in the breast just before a period It is not in itself painful, is a very frequent condition, and may be termed physiological

If, however, the condition progresses, the epithelialization of the ducts and acini spreads and begins to affect the larger ducts Moreover, as the heaped-up cells disintegrate, the acinar contents increase and distension into cysts occurs The type of cells begins to change and the epithelium which is being formed is swollen, feathery, and stains poorly In mazoplasia and in this cystiphorous variety a lymphocytic infiltration is seen Lymphocytes are also seen in the thyroid in nodular states, and are probably in both organs an attempt by the tissues to counteract the mal-involution by absorption of the degenerating epithelium Bergels¹⁵



FIG 292



FIG 293



FIG 294



FIG 295

FIGS 292, 293 —Sections of clinically normal breasts. These figures show the epithelial hyperplasia of mazoplasia. This is almost universal. The slight expansion of the ducts and acini in Fig 292 has progressed in Fig 293 to a precystic distension.

FIG 294 —Hyper-involution type of cyst with a single flattened layer of epithelium.

FIG 295 —Hypo-involution type of cyst showing papilliform projection of epithelium into the lumen (benign neoplasia).

FIG 296 —The same type of case as Fig 295, but showing at A the epithelial outgrowth becoming sessile and the disappearance of the connective-tissue element (pre-malignant).



FIG 296

states that they are always present where a physiological or pathological autolysis is in progress

There are two schools of thought with regard to cyst formation—one, which blames obstruction by plugs of epithelial debris, inflammatory processes, and proliferating connective tissue, and the other which regards them as due primarily to an epithelial hyperplasia. Cheate, who holds the latter view, points out that the strong-walled ducts alone may undergo cystic change, while the delicately-walled acini in communication with them are unaffected, whereas the reverse would occur were obstruction the cause. Moreover, where scars or large carcinomata affect the base of the nipple and mechanically obstruct all the ducts there is often a complete absence of dilatation of their peripheral branches and acini. Of the women in our series, 15 had had breast abscesses opened years before, but of these only 2 showed any nodularity whatever, and even these were not demonstrably cystic, in spite of the scarring and subsequent duct obstruction which must have occurred.

It is held in this article that all cysts are due to faulty involution, frequently they are found to have the appearance of a true retention cyst, the lining consisting of one flattened layer of cells with no evidence of cellular proliferation anywhere. These are to be regarded as areas of hyper-involution (*Fig 294*). Cysts with an epithelial lining are the result of an incomplete involution (*Figs 292, 293, 295*). The next stage from cystic formation is the supervention of epithelial neoplasia which may occur in the more active parts—that is, in the smaller cysts, where the epithelium is not degenerated so extensively. This epithelial neoplasia is at first benign and appears as a papilliferous outgrowth into the lumen. Later still these epithelial outgrowths may become sessile and lose their connective-tissue element, the cells are de-differentiated and the picture begins to take on the characters of malignancy (*Fig 296*).

It does not follow that this change will occur in every cystic breast, it is the supervention of a different process—that of epithelial neoplasia upon one of epithelial desquamation—whether *post hoc* or *propter hoc* only further research can decide. The facts that the fluid in cysts contains irritant substances such as urea, that experimental tar carcinomata of the breast in rodents show the stages of preliminary desquamation, and that there is evidence that cellular growth is stimulated by autolysis of cells apart from any irritant substances produced by such autolysis (Drew¹⁶), are reasons for believing that cystic changes in the breast may lead in certain cases to malignancy.

Cheate states that 20 per cent of all carcinomata originate in cystiphorous desquamative hyperplasia. Five cases of the women examined by us had had cysts removed locally at times varying from two to ten years previously. Four of these had completely normal breasts, the remaining one was in hospital for removal of a cyst which had developed in another portion of the same breast.

THE INVESTIGATION

The 500 cases which formed the material for our investigation ranged from the ages of 10 to 80 and were divided into age groups of 10-20, 20-30, 30-40, 40-50, and 50 upwards. Nodularity of the breast in greater or less degree, and showing all the clinical signs indicative of mastoplasia, was found in 144 cases—a

percentage of 28.8. Of these 144 cases, 66 were single and 78 married (Of the latter 78, 40 had suckled one or more of their children) The percentage incidence of nodularity in the various groups was as follows —

10-20	28.6
20-30	33.0
30-40	32.0
40-50	33.0
50 upwards	21.0

These figures show in a striking manner the universality of the nodular breast (Fig 297). It is, roughly speaking, equally common in the married and unmarried, and the three decades between 20 and 50 show a remarkable equality in the incidence of the condition—namely, 33, 32, and 33 per cent

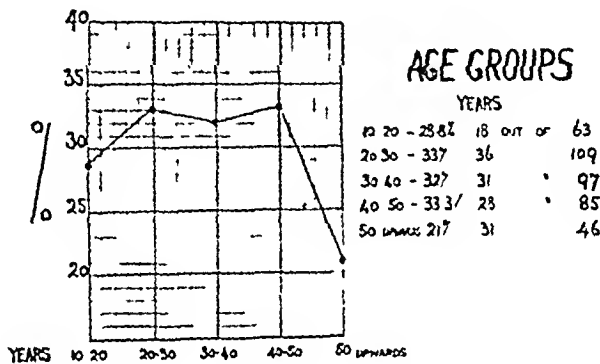


FIG 297 —Graph showing incidence of nodularity in various age-periods

Nearly all of those who were between puberty and the menopause showed signs of ovarian hypofunction, their periods being painful and scanty, and were of the asthenic, chronically unhealthy type. Before puberty it does not occur, and after the menopause an involutionary cure is brought about. Brodie,¹⁷ in his original description of the disease in 1846, says "To complete the history of the disease as it first shows itself I may add that the general health is unaffected, that the patient complains of no pain unless it be that in some instances there are disagreeable sensations that are apt to arise wherever the attention is anxiously directed to any one part of the body." The truth of this statement was borne out by the fact that of the 500 cases examined, only 46 complained of pain in the breast, and that of these 46, 20 (nearly half) had no signs of mastoplasia or cystic change whatever. Moreover, in the 26 who had nodular breasts which *were* painful, it was found that the pain bore no relation to the degree of nodularity found.

By far the majority of nodular breasts—98 out of 144 (68 per cent) made no complaint whatever of pain, and the conclusion became obvious that pain was not a feature of the condition, but was, as Brodie suggested, "a disagreeable nervous sensation arising when the attention was anxiously drawn to that part", and I cannot help feeling that in the past many breasts have been removed not so much on the clinical findings and judgement of the surgeon as on the demands and anxiety of the patient.

That the definitely cystiphorous stage is relatively uncommon was shown by the fact that only 20 out of the 500 had demonstrable cysts in their breasts. I have no doubt, however, in view of the pathological findings, which I refer to later, that in many of the 144 cases of nodular breasts small clinically unrecognizable cysts would have been found if the affected portions had been excised and sectioned.

There were 33 cases of carcinoma of the breast in the series of these, 6 showed nodularity or cystic change elsewhere in one or other breast, giving a percentage of 18, which is a figure approximately the same as that given by Cheatle for carcinomata arising in cystic breasts. It is of course possible, however, that the nodularity in these cases was secondary to the malignant growth.

The average age of patients with non-cystic nodular breasts was 36, in those with cysts it was 40, and in the malignant cases 50.

PATHOLOGY

A series of sections of breasts of female post-mortem cases with no history of breast disease were provided by the Pathological Department of the Queen's Hospital, Birmingham, and were examined. The following conclusions were arrived at—

1 **Mazoplasia**—It was expected in view of the clinical frequency of nodular breasts that mazoplasia would be seen in a large percentage of these sections. The histological picture of the condition was actually found in almost every instance (15 out of 17) whether cysts were also present or not.

2 **Cysts**—If mazoplasia, a very common condition, is the first stage of a process leading to clinically obvious cyst formation, as is held in this paper, then an intermediate stage where cysts are demonstrable microscopically only should be found with a frequency less than that of mazoplasia but greater than that of clinical cystic disease, and this in effect was what these sections showed. Out of 18, 8 showed cysts larger than that of the average glomerulus. Moreover, in these sections there were always to be found areas of mazoplasia, and all stages of cyst formation from the slight terminal distension of ducts and acini seen in the latter, to those visible to the naked eye, and showing all the characteristic cytology of cystiphorous desquamative hyperplasia. In short, the difference between mazoplasia and the cystic breast seemed to be in degree and not in kind.

Histologically some cysts are true retention cysts with no evidence of epithelial hyperplasia. These are to be explained as a localized area of hyper-involution. In two cases papilliform projections into cysts were seen. These epithelial proliferations were hyperplastic and not neoplastic, the cells being equal and typical.

A series of sections of portions of cystic breasts removed surgically were examined, and in some cases (*see Fig 296*) the benign neoplasia seen in the walls of cysts where cellular activity was greatest showed a different picture. The connective-tissue element of the papillæ had decreased, and cell masses were formed. The cells were not irregular and atypical, but the possibility of such a condition being precancerous cannot be ignored, the post-mortem cases, however, show

that unsuspected cysts are so frequent that the cystic state cannot be generally condemned. Cases should be treated as premalignant only when microscopical section following biopsy reveals the picture just described.

TREATMENT

To test the clinical application of the theory of the endocrine causation of painful nodular breasts a small number of cases treated by Mr Hugh Donovan as out-patients were followed up, and with his permission are included in this paper. To these were added a few patients seen in the Casualty Department and treated there. In all they numbered 15, and the treatment was by the administration of ovarian residue for the last half of the intermenstrual period. In Mr Donovan's cases the procedure employed was that advised by Cheatle and Cutler—namely, to order the patient to take tablets of ovarian residue containing 5 gr three times a day. The tablets used were those made by Paynes & Burn. Recently, however, all patients have been treated with daily injections of the Parke Davis product known as Theelin, recommended by Professor Whitehouse, during the same period of time—2 c c, corresponding to 100 standardized Doisy Rat Units, were given at each injection. Twelve of these patients, of whom 5 had definite cysts, reported a remarkable improvement as regards pain, and there was a noticeable diminution of nodularity. In 3 cases all traces of knottness had disappeared. In the remaining 3 there was no improvement.

No direct control such as the injection of sterile water was employed in this series, but 9 of the patients had previously had courses of X-ray treatment without any benefit, clinically or symptomatically, being reported by any of them. This small series of cases in conjunction with the series published by Cutler and Whitehouse show that treatment by the œstrus-producing ovarian hormone has a definite place in the treatment of this perversion of breast physiology. It cannot, however, be expected to replace surgery where there is a gross cystic change. The treatment by puncture, aspiration of cysts, and subsequent ovarian therapy mentioned by Whitehouse must have a limited application.

CONCLUSIONS

1 Non-cystic nodularity of the breast is a very common condition and is due to a faulty physiology dependent upon endocrine disorder.

2 This non-cystic nodularity may in certain instances pass on to a cystic state and is always a precursor of such cystic state, and this condition of the breast in all its stages should be known by one name as 'mastopathia', first suggested by Aschoff and recently adopted by Whitehouse.

3 The condition commonly is not painful.

4 Unless cysts are obvious, treatment should be by ovarian residue aided by attention to the general health of the patient. In cystic states, local removal should be performed and further treatment decided by careful pathological examination, only a definitely malignant picture should call for a further radical operation.

Pre-malignant cases should be treated by a local amputation of the affected breast.

My thanks are due to the Honorary Surgical Staff of the Queen's Hospital, Birmingham, and to Mr Hugh Donovan especially for their advice and kindness in providing me with the material for the investigation which was carried out for the purpose of this paper

REFERENCES

- ¹ ASCHOFF, L, *Pathologische Anatomie*, 1913, II
- ² WHITEHOUSE, BECKWITH, *Surg, Gynecol and Obst*, 1934, LVIII, 279
- ³ CREIGHTON, C, *Contributions to the Physiology and Pathology of the Breast*, 1879 London
- ⁴ KEYNES, G, *Brit Jour Surg*, 1923-4, VI, 89
- ⁵ BERTELS, A, *Deut Zeits f Chir*, 1913, CXXX
- ⁶ LUKOWSKY, A, *Ibid*, 1921, CLXVII, 81
- RECLUS, P, *Rev de Chir*, 1883, V, 3, 709
- ⁸ CHEATLE, SIR G LENTHAL, and CUTLER, M, *Tumours of the Breast*, 1931 London
- ⁹ PARKES, A S, and BELLERBY, C W, *Jour of Physiol*, 1927, LVII, 301
- ¹⁰ LOEB, L, and HESSELBERG, C, *Jour of Exper Med*, 1917, VII, 285
- ¹¹ ROSENBERG, A, *Frankf Zeits f Pathol*, 1922, XXXI, 466
- ¹² POLANO, O, *Zeits f Geburtsh u Gynacol*, 1924, LXXXVII, 363
- ¹³ DIECKMANN, H, *Virchow's Arch*, 1926, CCXVI, 321
- ¹⁴ THOMSON, A P, *Lancet*, 1932, II, 229
- ¹⁵ BERGELS, S, *Die Lymphocytose*, 1921 Berlin
- ¹⁶ DREW, A H, *Brit Jour Exper Pathol*, 1923, IV, 46
- ¹⁷ BRODIE, SIR B C, *Lectures in Pathology and Surgery*, 1846 London

INTERSTITIAL RADIUM TREATMENT OF CARCINOMA OF THE BREAST: DESCRIPTION OF A RADICAL TECHNIQUE

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THE radium treatment of mammary carcinoma presents many diverse problems, as the method of treatment must take account not only of pathological anatomy, but also of the laws which govern the activity of the therapeutic agent. No uniformity of technique has yet been achieved by those who practise this branch of therapy. Various combinations of surface and interstitial methods have been, and are being, tried. The whole tendency in modern radium practice is away from interstitial methods towards external radiation. Unfortunately, mammary carcinoma does not lend itself to surface radiation by radium to any satisfactory extent. Short of telecurietherapy, it is not practicable to deliver a high dose throughout the potentially affected tissues, including the complete axilla and the internal mammary zone. In the present state of our knowledge, therefore, we must rely basically on interstitial methods. Nevertheless, it is probable that the ultimate choice in radiation therapy will be a combination of deep X rays and interstitial radium therapy, and already Pfahler, Kaplan, and others have formed favourable impressions of this procedure. Maximum efficiency of such a technique depends, however, upon the thorough exploration of these methods individually, so that an accurate assessment of the limiting possibilities of each may be made. The present communication deals entirely with the interstitial aspect of this problem.

Many interstitial techniques have been evolved. In this country and in America that published by Keynes has become almost standardized, and it is too well known to any who are interested in this subject to require description here. The method is open to criticism, as are all others to-day, but it has justified itself by the results achieved. These cannot be questioned, and some of Keynes's latest figures are convincing evidence that this method must hold a definite place in the treatment of cancer of the breast.

Kaplan has employed a modification of Keynes's technique which does not, however, appear to improve upon the original. Perhaps his most notable modification is the use in some cases of very long needles—up to 9 cm., with filtration equivalent to 0.5 mm. Pt.—inserted in relation to the breast and into the axilla. A similar modification is employed by Cutler, and by Lamont and Morrison in Glasgow. Souttar, Kaplan, and Lee and Pack have employed gold seed implantation as a method. Failla and Lee and Pack use capillary gold tubing containing emanation, cut to suitable lengths. Meland's technique resembles closely that of Keynes, and Monchaux uses a similar distribution except in relation to the breast itself, where he employs two concentric circles of needles. Various other modifications of Keynes's technique, and combination of it with X rays or surgery, or both, are in use.

Several criticisms are levelled at the interstitial method. Murdoch and Simon,

admitting that needling is capable of delivering a greater amount of actual energy to the tissues than can be delivered by surface methods, nevertheless condemn the procedure on the grounds of impossibility of attaining homogeneity. Such criticisms miss the fact that what is required is not homogeneity—admittedly impossible—but attainment throughout the whole area of a minimum intensity which will be lethal for the tumour cells, with a maximum intensity which will be insufficient to interfere with the vitality of the normal tissues.

Regaud admits that needling is a simple and efficacious way of dealing with the primary growth in the breast, but has given it up for the fundamental reason that he believes it to be dangerous as being productive of metastases. Such a statement demands attention, but would appear to lack actual proof. Lee, Pack, Quimby, and Stewart state that in a series of 130 cases treated by interstitial methods they have not seen evidence of dissemination from this cause.

One of the major difficulties is to secure a satisfactory distribution of radium foci. This article describes a technique which is designed to surmount this obstacle. Theoretically it is hardly possible to contradict Souttar's claim that gold seeds are the nearest approach to the ideal vehicles for attaining relative uniformity of radiation. Lee and Pack, however, deal with this matter in an interesting article on measured tissue dosage in the radiation of mammary carcinoma. With reference to the relative claims of gold seeds and of linear foci they have shown in their experience that the practical difficulties of distribution of gold seeds outweigh theoretical considerations, and have adduced not only radiographic but also histological evidence in favour of their contention. Cutler and Martin discuss this question also, and decide in favour of needles. Irradiation by linear foci would, therefore, appear to be the method of choice for interstitial work.

All the interstitial methods published to date are incomplete. In 1931 Birkett criticized Keynes's method on this ground, admitting, however, that the final issue could lie only with the results. As mentioned above, Keynes's recent publications have supplied a satisfactory answer up to a point. Satisfactory results *have* been obtained, but it is a question of improving upon them. If methods admittedly amenable to improvement are capable of giving useful results, better results may be expected from a successful revision of the methods. Where Keynes's technique falls short, the other techniques are equally faulty. All are subject to criticism on two main grounds: (1) Incompleteness of radiation as regards the upper axilla, supraclavicular area, and internal mammary zone, and (2) The faulty physical conception of the distribution of radium foci.

In spite of these criticisms, Keynes with his standard method was able in 1929 to show initial good results in 67 per cent of cases in Grades I and II and in 46 per cent of cases in Grade III, with 30 per cent of apparent cures taking all groups together. At the Meeting of the British Medical Association (Dublin, 1933) he stated that 7 out of 25 (28 per cent) inoperable cases treated had survived for five years without disease.

Souttar's experience has been such as to convince him that the surgeon can offer radium implantation as an alternative to surgery without fear of prejudice to the interests of the patient.

Lee and Pack consider that interstitial implantation can be safely advised when operation is contra-indicated, and that it is justifiable to employ the method in primary operable cases.

RADICAL TECHNIQUE

In comparing radiological with surgical history, it is relevant to note two points (1) The striking improvement in surgical results ushered in with the introduction of the radical (i.e., surgically complete) technique in amputation, and (2) The strikingly poor results in cases treated by operations of the incomplete type, as shown in such publications as those of Greenough and Lane-Claypon. If the radiological parallel could be achieved, it is felt that results would improve in a manner equally impressive. Using Keynes's technique as a starting-point, work has been going on in the Holt Radium Institute, Manchester, for the last two years or so, in the gradual evolution of a technique which is now brought forward with the claim that it is a radical (i.e., radiologically complete) implantation. It is introduced as being surgically and radiologically sound, and as having been tested thoroughly as to practicability over a considerable number of cases to date.

The modifications of existing methods involved differences in dosage, in volume-irradiation, and in anatomical considerations so essential as to bring the method within the confines of the experimental. The technique has therefore been developed on inoperable cases, mostly well beyond the bounds of the most optimistic surgery, many beyond the bounds of reasonable radiological expectation. In view of practical experience of the method, however, and of the good results obtained by other implantation methods, it is felt that its application now to operable cases in certain circumstances is entirely justified, and that it must be considered seriously in those cases of border-line operability in which the results of surgery alone are so disappointing. It is not, however, pressed at present as a method of choice to be used to replace surgery with its known results.

The technique as developed in Manchester is as follows —

1 **Breast.**—According to the size of the breast (tumour *qua* tumour is disregarded) one or more layers of needles are inserted from medial and lateral sides, in such a way that the needles of the deepest layer, undercutting the breast, do not meet in the centre, and that any subsequent more superficial layers approximate gradually more closely until the effect achieved is that of a cone of radio-active foci with a central zone adequately irradiated but not containing actual needles. The peripheral ends of the needles are crossed at the base of the cone, and occasionally in the subareolar area one or more needles are inserted at 90° to the deeper planes to close the apex of the cone (Fig 298). Such peripheralization of radium foci avoids the high central intensity, falling rapidly towards the periphery, inevitable when any volume of tissue is implanted in a uniform or in a cart-wheel manner. The needles used are of a content of 3 mgrm, active length 4.5 cm screened by 0.5 mm Pt, and of 2 mgrm, active length 3 cm, with similar filtration.

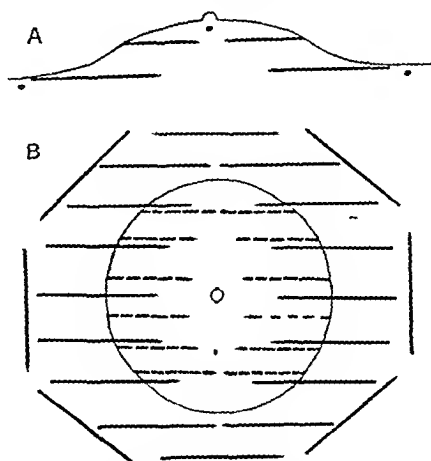


FIG 298.—Diagrammatic representation of breast implantation in three planes. A Elevation, B Plan. Deepest layer of needles continuous, other layers dotted.

2 **Axilla**—For purposes of radiation the axilla is divided into two separate areas—base and apex. As in the case of the breast, peripheralization of the radium foci is aimed at

a Base—Depending upon the patient's build, a variable number of needles, usually two, three, or four, is inserted along each of the four walls of the axilla. The medial ones run in close relationship to the chest wall, the lateral in close relationship to the great vessels, the anterior in the interpectoral plane, and the posterior in relation to the anterior surface of the subscapular muscle. The general effect is that of a cylinder of needles, as shown in *Fig 299*. All these needles are of 3-mgrm content with active length of 4.5 cm and with 0.5 mm Pt filtration.

b Apex—Two or more needles are inserted parallel to the clavicle, running deeply in the pectoralis major close to the costocoracoid membrane. Two or more of the same type are inserted through the pectoral musculature at 90° to the chest wall at the level of the upper axilla, forming tangents to the ribs, the upper one calculated to pass just below the line of the vessels. These two sets of needles form a bracket round the antero-lateral aspect of the upper axilla. To complete

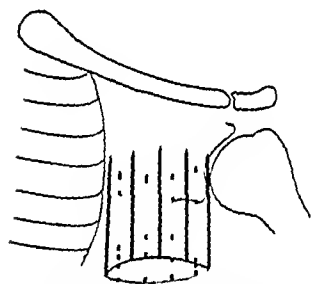


FIG 299—Diagrammatic representation of implantation of base of axilla, showing cylindrical effect

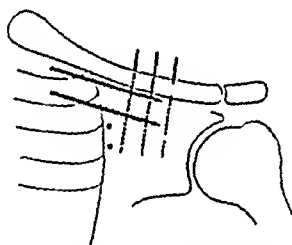


FIG 300—Diagrammatic representation of implantation of apex of axilla. Retroclavicular needles dotted

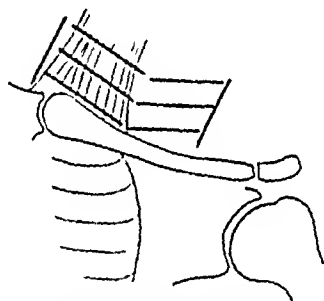


FIG 301—Diagrammatic representation of implantation of supraclavicular area

the bracketing of the upper axilla, two or three needles are inserted from above the clavicle, and run down into the apex of the axilla. The most medial needle is inserted immediately lateral to the third part of the subclavian artery. Anatomical necessity determines that these needles run along the postero-lateral aspect of the apex of the axilla. All the needles are of 3-mgrm content, 4.5 cm in active length, and filtered with 0.5 mm Pt. X-ray photographs demonstrate that a satisfactory distribution of these needles can be achieved. Up to the time of writing no damage to important vessels or other structures has occurred. The distribution is indicated in *Fig 300*.

3 **Supraclavicular Area**—Three needles are inserted deeply into the substance of the sternomastoid muscle, directed lateralwards from its medial border. Three others are inserted in the opposite direction, running across the posterior triangle to meet, or almost meet, the points of the former. A vertical needle is inserted at the level of the heads of each of these sets of needles in order to increase the field of irradiation to a satisfactory extent. In this way the two sets of needles are made to form a bracket round the lower part of the great vessels at the root of the neck. Several of the X-ray photographs demonstrate this, and *Fig 301*

illustrates the technique. The needles used in this field are of 1 33-mgrm content, of 2 cm active length, and are filtered with 0.5 mm Pt.

4 **Mediastinum**—Using any suitable seed-introducer, such as Souttar's gun, three gold seeds are inserted into the anterior mediastinum through each of the upper four intercostal spaces. Usually seeds of 1.5 mc strength and screened with 0.5 mm gold are used. The cannula is inserted obliquely into the intercostal space until the edge of the sternum is encountered. From that as starting-point it is introduced under the edge of the sternum and under the edge of each of the adjacent ribs in turn, one seed being deposited at each of these points, so that there is a triangle of seeds in the mediastinal area opposite each of the upper four intercostal spaces. *Fig 302*, prepared by Mr H M Parker, Physicist to the Institute, indicates the field of intensity round such a distribution of gold seeds as opposed to that round a single needle of 1 33 mgrm and 2 cm active length.

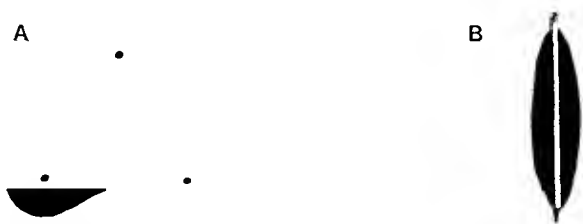


FIG 302—Mediastinal technique. A, Three gold seeds, 1.5 cm apart, each = 1.5 mc, filtration = 0.5 Au. Time = ∞ . Area receiving 6000 r units or more is shaded. B, One 1 33-mgrm needle, active length 2 cm, filtration = 0.5 Pt. Time = 7 days. Area receiving 6000 r units or more is shaded (same scale as A). (By Mr H M Parker.)

The advantage of the former over the latter is apparent, and becomes even more obvious when it is considered that the seeds lie in the plane of the mediastinum, whereas a considerable part of the active length of any mediastinal needle must always be superficial to this plane. Indeed, in obese patients the slant of the mediastinal needle becomes of necessity very considerable, and its insertion at all a matter, in some cases, of extreme difficulty. Where no gold seeds are available, however, needling is employed as described by Keynes, but using when possible two needles in each interspace instead of one.

FEATURES OF THE NEW TECHNIQUE

It will be noted that the main features of the technique as described are —

- 1 The radiation of a large volume of tissue in continuity. In so far as such a feature can be illustrated, this is seen in some of the X-ray photographs.
- 2 The application of the allied principles of peripheralization of radium foci and of cross-fire radiation. These features also are illustrated to some extent in certain of the accompanying X-rays.
- 3 The new approach to the problem of mediastinal lymphatic area involvement.
- 4 The new approach to the radiation of the upper axilla so that an unbroken volume of radiation extends from breast to supraclavicular area.
- 5 The extended radiation of the supraclavicular zone.

Stereoscopic X-ray films bear out the contention that it is possible to radiate the breast and its lymphatic areas in a satisfactory manner. Already the study

of such films has led to a number of modifications and improvements, and so valuable has been the information thus secured that any new departure in technique is now critically examined by this means. This method of examination cannot be too strongly recommended to any who may consider the adoption of a technique such as has been described.

While non-stereoscopic films are somewhat confusing, and at times a little difficult of interpretation, they afford, nevertheless, some indication of the various features described above. A number of these are reproduced herewith, and serve to illustrate the gradual development of the technique described, as well as its more distinctive features. *Fig 303* shows an early phase, roughly corresponding to the



FIG 303—X-ray illustrating development of radical technique. Early stage. Conforms roughly to Keynes's technique, but with omission of infraclavicular needles.



FIG 304—X-ray illustrating development of radical technique. Later stage. Note (1) Two antero-posterior needles in upper axilla, (2) Two infraclavicular needles, (3) 'Hollow cone' in plantation of breast.

Keynes technique, but with omission of infraclavicular needles. The presence of these last would have improved the radiographic effect, but would have done little to radiate the upper axilla. This problem of the upper axilla called for serious consideration, and *Fig 304* shows an early attempt at its solution. Satisfaction was experienced, however, only with the introduction at a later date of retro-clavicular needles, as shown in *Figs 305* and *306* and also in subsequent prints. Minor modifications in supraclavicular and mammary distribution followed, and can be seen in *Figs 306* and *308*. At this stage it was felt that a sufficient volume was being radiated in continuity, but that the radiation of the internal mammary zone—by needles inserted in the intercostal spaces—was inadequate. The final step in the evolution of the technique was devised to meet this criticism, and is illustrated in *Figs 307* and *308*, where three radon seeds have been inserted via each of the upper four intercostal spaces, and are lying in a plane posterior to the deep aspect of the sternocostal junctions.



FIG 305—X-ray illustrating development of radical technique. Later stage showing (1) Supraclavicular bracket region of great vessels, (2) Complete axillary implant, with retroclavicular needles.



FIG 306—X-ray illustrating development of radical technique. Later stage showing (1) Supraclavicular bracket region of great vessels, (2) Complete axillary implant, with retroclavicular needles, (3) P. irradiation, (4) Duplication of intercostal needles.

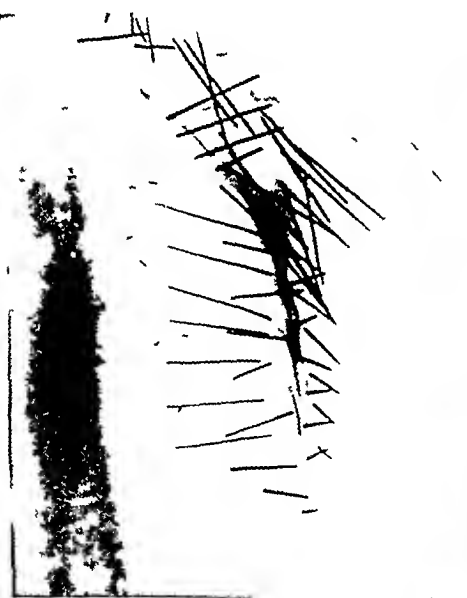


FIG 307—X-ray illustrating development of radical technique. Later stage showing previous features and illustrating the gold seed technique for the internal mammary zone. One of the antero posterior needles of the upper axillary group can be seen end-on.



FIG 308—Illustrating development of radical technique. Similar to Fig 307, but with peripheral outlining of breast field.

DURATION OF TREATMENT, END-RESULTS, ETC

Duration of Treatment, etc—The needles are left in position for seven days as a rule, but this period has been empirically arrived at, and there is room for experimental determination of the optimum time-factor. Alteration of the time-factor could, of course, be carried out, if desired, without modification of the total dose, as the total number of needles could be increased or diminished, or the linear intensity modified, without departure from the distribution outlined above.

During treatment the arm on the affected side is kept at rest with the humerus abducted to a right angle. The object of this is to prevent undue reaction on the skin of the axilla by maintaining the disposition of the axillary needles. Pillows are not satisfactory for this purpose, and a suitable splint is worn. The reaction usually proceeds to the extent of a mild radio-epidermitis in one or more areas.

Definite blood changes are produced as the result of the radiation of such a volume of tissue. A sharp fall in the white-cell count is an invariable accompaniment of the treatment. Dr Goodfellow, who has been working on this problem for some time, informs me that an immediate leucocytosis—in most cases confined to the polymorphonuclear cells—is followed by a steady fall in the number of white cells, most notable in the case of the lymphocytes.

End-results—As experience of distribution of radium foci and of total dosage accumulates, complete disappearance, not only of the tumour mass, but also of glandular deposits, becomes more and more frequent. Indeed, it can be stated that there appears to be an optimum dosage at which even the largest primary and secondary masses, provided that the tumours are of sensitive type, undergo complete resolution, leaving no trace of their presence. When such dosage is not achieved, residual indurations, sometimes very vague and indefinite, sometimes more localized, may occur. It seems from the consideration of cases in one's own experience that such residues can occur as the result of departure from the optimum dosage in the direction either of insufficiency or of excess. That some workers have demonstrated in such masses cancer cells in apparently vital condition is no proof that the same would hold after more appropriate amounts of radiation. It must be the experience of all radium workers that high dosage in any tissue is liable to produce definite induration. Such an effect is probably peculiarly liable to occur in the breast, the areolar tissues of which appear to lend themselves readily to some form of proliferative fibrosis under radiation.

The complete and striking disappearance of affected lymph-glands in many cases is interesting, and disappearance of these seems to be more consistently achieved than of the primary growth itself.

The work described in this article was initiated in the latter half of 1931. For reasons previously stated the treatment was confined to very advanced cases, except in occasional instances when other factors, such as the general condition of the patient, had to be taken into account.

In the course of 1931 and 1932, 53 cases were treated, starting on the model of Keynes's technique and gradually evolving therefrom. During this period the policy was consistently adopted of referring all apparently operable cases for surgery, and of treating earlier cases only if there were definite surgical contra-indications. Of the 53 cases treated, 17 (32 per cent) are alive over one year.

- LANE-CLAYTON, *Cancer of the Breast and its Surgical Treatment*, Ministry of Health, London, 1924
- LEE, B J, *Ann of Surg*, 1928, July, 26
- LEE, B J, *Amer Jour Surg*, 1930, NS VIII, 134
- LEE, B J, *Proceedings International Conference on Cancer*, London, 1928, 1931
- LEE, B J, *Amer Jour Roentgenol*, 1932, April, 547
- LEE, B J, and PACK, G T, *Bull Memorial Hosp, N Y*, 1929, 1, 67
- LEE, B J, and PACK, G T, *Acta Radiol*, 1931
- LEE, B J, PACK, G T, QUIMBY, E H, and STEWART, F W, *Arch of Surg*, 1932, XLIV, 339
- MARTIN, C L, *Amer Jour Roentgenol*, 1932, Feb, XXVII
- MELAND, O N, *Ibid*, Aug, XXVIII, 223
- MONCHAUX, C DE, *New Zealand Med Jour*, 1932, Dec, 383
- PACK, G T, *Amer Jour Roentgenol*, 1932, XXVII, 532
- PFÄHLER, G E, *Ibid*, 497
- REGAUD, C, *Arch Inst du Radium de l'Univ de Paris*, 1929
- REGAUD, C, *Bol Soc Obstet y Ginecol de Buenos Aires*, 1930, IX, No 14
- SCHREINER, B F, *Ann of Surg*, 1931, XCIII, 269
- SOUTTAR, H S, *Brit Med Jour*, 1933, 1, 813
- WEBSTER, J H D, *Brit Jour Radiol*, 1926, XXI, 59
- WEBSTER, J H D, *Lancet*, 1928, II, 63
- WEBSTER, J H D, *Brit Med Jour*, 1932, II, 47

DIAPHRAGMATIC HERNIA

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(Being an Arris and Gale Lecture, delivered at the Royal College of Surgeons of England on February 23, 1934)

ALTHOUGH instances of diaphragmatic hernia not due to trauma have long been recognized, the condition is still sufficiently rare, of sufficient gravity, and there remain so many debatable questions associated with it, to make it desirable that the investigations carried out in a series of cases should be recorded

Sir Arthur Keith¹ and Basil Hume² have written on the embryology, and Woodburn Morison³ has reviewed the history and discussed the radiology of the subject. A. F. Hurst⁴ and his colleagues have published most valuable clinical records. The late R. P. Rowlands, Salisbury, Lake, and others, have each published a case operated upon with cure of the patient. The total number of published cases operated upon in this country would appear to be small, while in America C. A. Hedblom⁵ had published as long ago as 1925 a series of 9 cases operated upon by him, and Stuart Harrington⁶ a series—including 13 of traumatic origin—of 45 cases from the Mayo Clinic in 1931. It can scarcely be that the proportion of cases in the population should be so much higher in America. We must rather believe that, in patients with doubtful symptoms, the search is more thorough in the big American clinics, and that when a diaphragmatic hernia is found to be the cause of disability the patient is more frequently offered, or asks for, surgical relief.

A number of cases in infants have been reported in British journals. Formerly most of these were discovered post mortem, after a short and distressing struggle for life had ended. The death-rate in infancy is high because the extent of the defect in the diaphragm is in some cases so extensive that it is scarcely compatible with life. This aspect of the question has been admirably dealt with by N. R. Barrett and C. E. W. Wheaton.⁷ When the defect is not such as to terminate life in infancy the individual may lead a normal life until a later period, when symptoms may appear more or less gradually. The patients upon whom the investigations set out in this paper have been made have come under my observation at this later period of life.

As the literature of the subject has been frequently and recently reviewed and is readily accessible, it would seem to be most useful to give my personal experiences.

My series is made up of 25 patients in 11 of whom the œsophagus was of normal length, with part, or the whole, of one or more abdominal organs herniated into the thorax. In the remaining 14 patients the œsophagus was congenitally short and a portion of the stomach was situated in the thorax. Of the former 11, 8 were carefully investigated clinically and radiologically and then operated upon. The latter 14 patients—with short œsophagus—were investigated on the whole less completely because in most of these cases the investigations were not completed

by operation. Apart from the relief given to the patient, operation has afforded the opportunity of confirming diagnosis and verifying the anatomical conditions present. The "pathology of the living" will always remain of interest and value, for the patients are alive and available for subsequent observation. Even though patients with a short œsophagus may not in the majority of instances, or in the present state of our technical skill, be so suitable for operation, observation of some of these patients over a period of years has given valuable information, so that we become more familiar with the behaviour and prognosis of this by no means rare condition.

CLASSIFICATION

A number of classifications have been published. Perhaps the time has scarcely arrived for one which would be universally acceptable. If each investigator arranges his cases in the manner that best illustrates his findings, a suitable classification will emerge. For these reasons I have arranged my cases according to the site at which the abdominal organ, or organs, entered the thorax. As none of these herniæ has followed any known injury, I have assumed that they are congenital. Seeing that imperfections of development are so well recognized in other parts of the body, constituting such deformities as hare-lip, cleft palate, spina bifida, imperforate anus, and hypospadias, it would be strange if a structure so complicated in its origin as the diaphragm did not sometimes fail to develop perfectly, and, furthermore, if fusion is less than perfect, the strains to which the diaphragm is subjected in early life (for example, during the spasms of infantile crying) are likely to make manifest any frailty. While it cannot be denied that some of these herniæ may have been acquired during life—muscle fibres torn in the spasms of whooping-cough with hæmorrhage followed by fibrosis and stretching—embryological evidence obvious by skiagraphy or revealed at operation shows that some at least are congenital.

The sites of the hernial orifices in the diaphragm in my series are as follows —

	Cases
I Retrosternal (synonyms—Parasternal, Foramen of Morgagni)	1
II Left dome	2
III Costo-vertebral region—left (synonym—Hiatus pleuro-peritonealis)	
(?) Non-development of left crus	3
IV Œsophageal region—	
1 Hernia diaphragmatica transversa. The œsophagus is of normal length, the anatomical hiatus is replaced by large defect (?) Due to non-development of crura	3
2 Para-œsophageal. The œsophagus is of normal length, the hiatus is anatomically normal but dilated	2
3 Short œsophagus, with partial or complete thoracic stomach	14

The description of the clinical types will be more readily understood if the development is considered. I am indebted to my colleague, Dr Woollard, Professor of Anatomy, St Bartholomew's Hospital, for the notes on embryology, and to his department for the drawings illustrating them.

DEVELOPMENT OF THE DIAPHRAGM

From our point of view there are three stages. In the first the coelomic space is continuous, until the growth of the embryonic organs subdivides it into pericardial cavity, pleural space, and peritoneal cavity. The pericardial cavity lies ventrally, is immediately in front of the septum transversum, and communicates

dorsally and in front (cranially) with the enlarging pleural space. The pleural space joins over the dorsal margin of the septum transversum with the peritoneal cavity. The channel of communication is called the pleuro-peritoneal canal (*Fig 309*) and the walls of the canal are the pleuro-peritoneal membranes. The septum transversum (*Fig 310*) is restricted to the ventral half of the embryo. The interval left between its dorsal margin and the back of the embryo is occupied by the canals just mentioned, by the developing œsophagus, and by stomach suspended in the median plane by the mesentery, and for a time by the growing lungs.

It is clear that if the pleuro-peritoneal canals should remain open through deficiency in the growth energy of the parts whose proliferation normally closes them, then the pleural and peritoneal cavities will not be separated completely from one another, and the

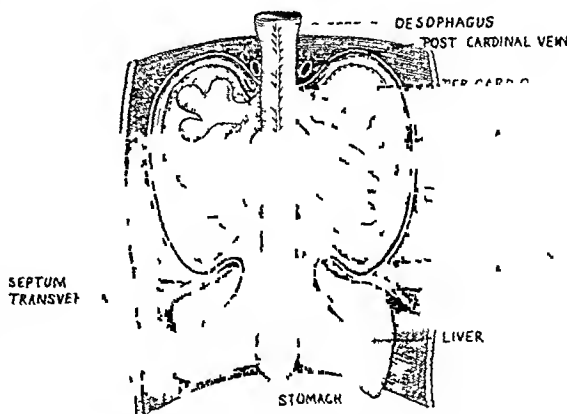


FIG 309—Showing pleuro-peritoneal canal

positive intra-abdominal pressure sooner or later will carry some or other of the abdominal organs into the thorax. This is one of the commonest types of hernia in infants, but if it occurs at this stage no sac is present, for the pleuro-peritoneal canal has not been closed. In the great majority of infants who die as the result of a diaphragmatic hernia in the first few hours or days following birth, and in

whom no sac is present, the hernia has probably occurred at this stage. This fact has given rise to the statement by Keith, Hume, and others that in herniæ in the pleuro-peritoneal region no sac is present and the infants rarely live. In my patients with hernia in this situation the patients were obviously alive and a sac was present, the explanation of the discrepancy is probably that the herniæ in my patients had occurred in the next—but very slightly later—stage of development.

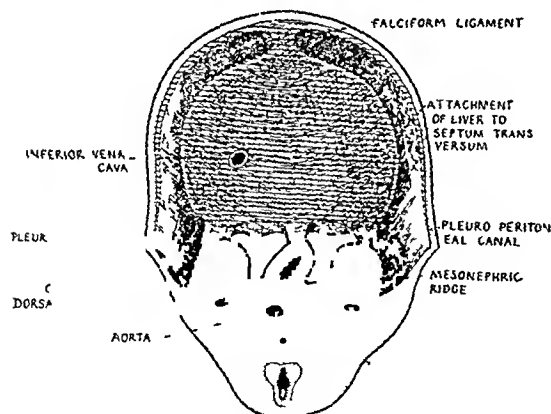


FIG 310—Pleuro-peritoneal canals in section

the second month of intra-uterine life and may be called the 'membranous stage'. During this period the pleuro-peritoneal canal is closed by the ingrowth of its own walls, proliferation from the mesentery of the gut, and the Wolffian ridge. Its position in the adult is indicated by the costo-vertebral triangle (*see Fig 310*). During this stage a true hernia of the diaphragm may occur, for the pleura and peritoneum are complete and have closed their respective cavities, and if any abdominal viscera are forced into the thorax at this time, a membranous sheet,

The second stage in the development of the diaphragm occurs during

which is the developing diaphragm itself, will be found interposed between their peritoneal covering and the pleura

The third stage in the development of the diaphragm occupies the third month. During this period the phrenic nerve preceded by the pre-muscle mass derived from the cervical myotomes spreads through the membranous diaphragm. The site from which the muscle spreads is indicated by the place where the nerve enters the diaphragm (*Fig 311*), and it grows gradually through the membranous structures, *reaching the dorsal margins last of all* (The significance of this is seen later in the description of *hernia diaphragmatica transversa*). In this way the whole membranous area becomes muscularized. Subsequently a change in the distribution of the muscle occurs and the central tendon of the diaphragm is developed.

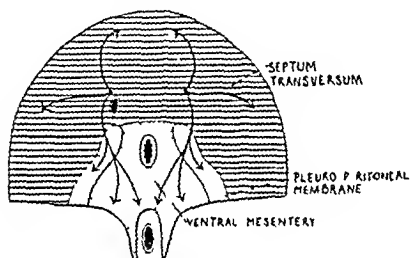


FIG 311—Showing where muscularization commences, and the directions of spread

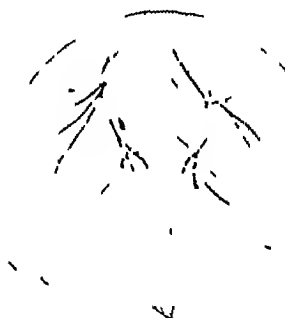


FIG 312—The muscular diaphragm and the phrenic nerve consist essentially of two parts—ventro lateral and dorso lateral

The growing muscle surrounds the cardia, forming the hiatus (*Fig 312*). The growth of the muscle may fail, leading to a deficiency in the muscular attachments of the diaphragm, although peritoneum and pleura separate the cavities.

In my series this growth deficiency had occurred bilaterally in the dorsal attachments in two cases and almost certainly in a third—the third I am not yet

able to confirm—and probably it had occurred in three others unilaterally. This condition really amounts to an absence of the crura together with the crural attachments, and the result is to leave a gap which permits the stomach with its peritoneal investments to slide up into the posterior mediastinum in the case of the bilateral defect, and into the left pleural cavity in the case of the unilateral defect, but as the oesophagus is of normal length it slides down into the abdominal cavity just as easily—this type has been called '*hernia diaphragmatica transversa*' (see *Figs 333-335*). Its significance will be seen later. I strongly suspect that the same type of defect occurring unilaterally

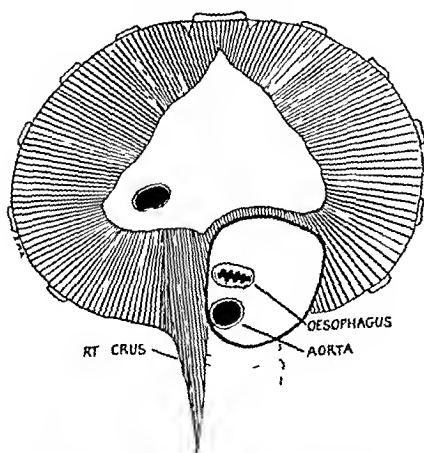


FIG 313—Defect in diaphragm arising from non-development of left crus, seen from below

(i.e., lack of development of the left crus) is the cause of the left costo-vertebral—the so-called pleuro-peritoneal—type when this is found in adults (*Fig 313*).

CLINICAL EXAMPLES OF THE VARIOUS TYPES OF DIAPHRAGMATIC HERNIA

I HERNIA THROUGH THE RETROSTERNAL ATTACHMENT OF THE DIAPHRAGM (PARASTERNAL, FORAMEN OF MORGAGNI)

Fig 314 is a drawing of the diaphragm from below showing the site of the defect which was the primary cause of the hernia. The aperture was 4 in wide and lay immediately behind the xiphoid appendix of the sternum. The patient was a man aged 29. He had suffered from indigestion and epigastric discomfort for five years. The symptoms occurred in attacks lasting from four to six weeks.

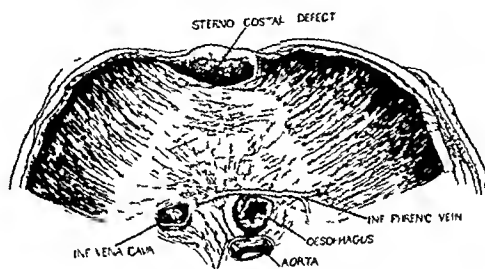


FIG 314—Showing site of retrosternal defect in diaphragm

He was always conscious of a "blown-out" feeling. He was relieved by passing flatus. He had become afraid to eat. Through pain and voluntary starvation he had lost 1 stone in weight and had been compelled to give up work. A barium meal or an enema (*Fig 315*) shows the termination of the ileum, the cæcum with the appendix, and what should be the ascending colon together with the transverse colon, in the thorax. The loop obviously enters and leaves by a narrow orifice. A second film (*Fig 316*) shows that no splenic flexure is present, the colon on leaving the thorax descends directly to the left iliac fossa. A third skiagram has been taken laterally (*Fig 317*). The colon had been filled with a barium emulsion, the patient placed in position, and the exposure made during the swallowing of barium emulsion. This outlines the oesophagus entering the abdomen in the normal position posterior to the pericardium, while the herniated viscera enter the thorax anteriorly immediately behind the lower end of the sternum. This patient's disability was due to nipping the segment of the alimentary canal and hampering its physiological activity.

The Sac.—In this case a sac was present. *Fig 318* shows the breadth of this and the extent to which the thorax was occupied. The sac and its contents occupied the anterior mediastinum, resting against the pericardium on the left and displacing the right pleura and lung backwards and to the right. The neck of the sac and its contents had spread the costosternal fibres of the diaphragm widely. After reduction the margins of the orifice were brought together easily by the sutures in the posterior half, but in the anterior half the gap was too wide and could only be closed by the aid of fascial graft sutures.

We cannot but be curious as to the period in the foetal life of this individual when he became foredoomed to the hernia. We have three pieces of evidence —

1 There was a defect in the diaphragm—this might have been developmental, but it might have been acquired through secondary atrophy, so that it does not help in determining whether the origin was pre- or post-natal

2 A peritoneal sac was present, therefore the peritoneum had shut off the abdominal from the thoracic cavity before its occurrence



FIG 315—Site of neck of sac. Termination of ileum, caecum, and portion of colon in the thorax

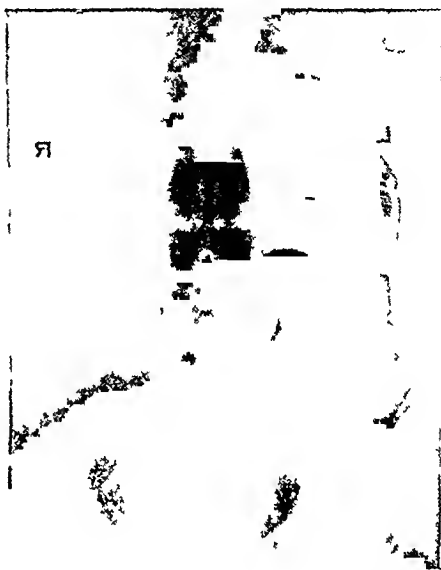


FIG 316—Showing absence of splenic flexure



FIG 317—Lateral view. 1 Contents of sac, 2 Oesophagus 3 Level of diaphragm



FIG 318—1 Terminal coil of ileum, 2 Appendix and colon, 3 Neck of hernial sac at site of the defect

3 The hernia had occurred before the rotation of the large gut was completed, for no splenic or hepatic flexure had developed, and the cæcum had never occupied its position in the right iliac fossa

The developmental story would be incomplete if I omitted to mention one post-operative sequence. As the sac and its contents were retrosternal, access was obtained by splitting the lower half of the sternum (*Fig 319*) and separating the two costosternal leaves after the abdomen had been opened by a comparatively short, high epigastric incision. At the end of an extensive operation it seemed expedient to fix the cæcum to the abdominal wall just below the liver, rather than

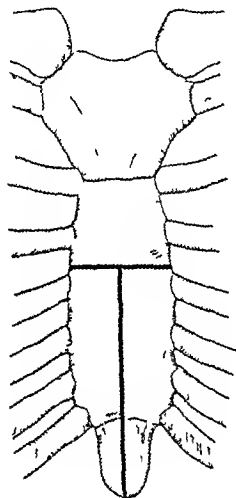


FIG 319—Line along which the sternum was sectioned



FIG 320—Skiagram showing that the cæcum has returned to its normal position

prolong the abdominal incision in order to attempt to place the cæcum in a more normal situation. *Fig 320* from a skiagram taken four weeks after the operation shows that by that time the cæcum had migrated to the right iliac fossa. During the twenty-nine years it had sojourned in the thorax it had never lost its developmental urge to occupy its normal position.

II HERNIA THROUGH THE LEFT DOME OF THE DIAPHRAGM

This hernia is actually through the substance of the diaphragm, and therefore to be distinguished from those through the left costo-vertebral angle, which—at any rate in two of my three cases—were really situated *behind* the diaphragm.

Fig 321 is a drawing showing the position of the defect. This is independent of any of the natural openings. I have two patients of this type. One was a woman of 52, whose serious symptoms were of only two or three months' duration. These rapidly increased in severity, and during this time she never became comfortable. She was afraid to take food. The pain was situated behind the sternum, in the epigastrium, and through the left thorax. Her face expressed anguish and this

distressed look never lifted. The second patient (*see Fig 325*) is a child of five years, who has periodic attacks of pain separated by weeks or months. His appetite is normal and food does not cause pain. The elderly woman I have operated upon, the parents of the child are considering operation, but when the attacks of pain pass they hesitate.

Skiagrams of the condition in the first patient show (*Fig 322*) (1) The œsophagus of normal length, (2) The actual cardia and small portion of the stomach in the abdomen—when outlined with barium it has the appearance of a filled champagne glass, (3) A very large middle portion of stomach in the left thorax.

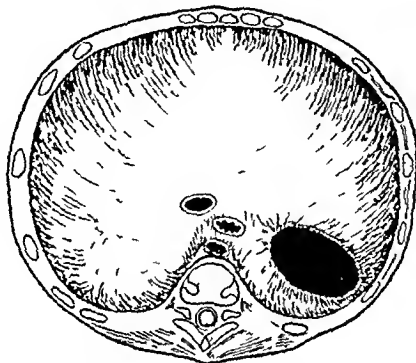


FIG 321—Drawing from below to show congenital defect in left half of diaphragm

distended with air, displacing the mediastinum with the pericardium and heart to the right. *Fig 323* shows the pylorus re-entering the abdomen. It can be seen that the left lung is compressed into the apex of the thorax.

Fig 323 with its explanatory sketch (*Fig 324*) shows beautifully the cardia entering and the pyloric portion of the stomach returning through the hernial orifice. This hernia was so extensive that it might have been mistaken for the condition known as eventration, or paralysis of the left phrenic nerve with elevation of the left leaf of the diaphragm, but when the skiagrams are scrutinized it is seen that the herniated structures are narrowed at the point of entry and exit, and that although the incarcerated portion of the stomach is so large, both the cardia and the pylorus are fixed in the abdomen. This would not occur with eventration. The points of entry and exit are away from the costo-vertebral angle and imply the position of the orifice.

The skiagrams cannot show, but operation revealed, that (1) No peritoneal sac was present, (2) Dense adhesions bound the stomach to the margins of the orifice, (3) Firmly organized adhesions bound the stomach to the compressed lung, (4) The stomach was rotated on its axis and the spleen was above the diaphragm, (5) The entire circumference of the orifice consisted of thick muscle.

The absence of a peritoneal sac implies that the defect had occurred before the membranous subdivision of the cœlomic space into pleural and peritoneal cavities, but the defective diaphragm had subsequently become completely muscularized up to the margins of the defect. The actual hernia must have occurred in the first stage of the development of the diaphragm. The condition present in this patient makes this an appropriate place to discuss the question of surgical access.

DIAPHRAGMATIC HERNIA

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FIG 322 — The œsophagus is seen entering the cardiac end of the stomach which is directed cranially. The main part of the stomach is situated in the thorax and is filled with gas. This has displaced the heart to the right (shadow seen on the left of the film).



FIG 323 — The œsophagus enters the stomach in the abdomen. The stomach at once enters the thorax. A narrow line of barium indicates its exit from the thorax, the pyloric portion of the stomach being in the abdomen. The aperture in the diaphragm is shown in Fig 321.

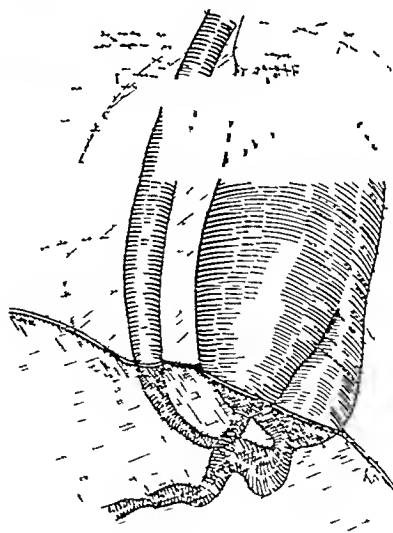


FIG 324 — This drawing explains Fig 323. It shows the adhesions between the stomach and the compressed lung. It is an exact reproduction of another film, with the adhesions drawn in.

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The Approach—Some surgeons favour the abdominal approach, others the thoracic. In this patient the adhesions of the stomach to the base of the lung, which was compressed into the apex of the pleural cavity, would have prevented withdrawal of the stomach through the orifice from the abdomen, nor could the spleen, which was adherent in the thorax, have been reduced. On the other hand, dense adhesions fixed the stomach in the neighbourhood of both cardia and pylorus to the margins of the orifice, and these would have prevented replacement of the stomach if a thoracic approach alone had been used. It was therefore necessary to open both the thorax and the abdomen. The great dilatation of the stomach would have prevented its replacement without deflation. After insertion of a pursestring suture, the stomach was emptied of its gas by a suction apparatus attached to a trocar and cannula. After the replacement of the stomach below



FIG. 325.—Hernia of transverse colon

the diaphragm, and repair of the hernial orifice, I felt sure that the patient could not for some time be happy with a stomach of this size in the abdomen. In patients where the stomach has been housed in the thorax for a long time the abdomen has adjusted itself to its absence. I had learned from patients upon whom I had operated previously that when the abdomen is suddenly asked to make room for an unaccustomed stomach—and a greatly distended one—much discomfort is induced, and this was the biggest stomach I had seen. After closing the thorax I therefore performed a gastrostomy, not for the purpose of feeding the patient but to give temporary exit to its gaseous contents. This manœuvre was justified, the patient being free from this discomfort after operation. Skiagrams following operation show the left lung completely expanded, and the stomach situated below the diaphragm.

The second patient with a hernia in this site is the child of 5 years. *Fig 325* shows the condition with part of the colon in the thorax. The attacks of pain only occur at intervals of some months. They are undoubtedly due to temporary obstruction of the colon. The child has not been operated upon. He is living in danger of an acute obstruction.

III HERNIA THROUGH THE COSTO-VERTEBRAL ANGLE (LEFT)

The third anatomical site is through the costo-vertebral angle of the left side (*Fig 326*). For reasons to be given later I consider this a more accurate designation than 'pleuro-peritoneal hiatus' or 'foramen of Bochdalek', at least in adult patients with a peritoneal sac present.

Screen examinations of two of the three patients of this type showed an œsophagus of normal length, and in the third this was proved to be so at operation. Other findings are —

- 1 A portion of the stomach and transverse colon may occupy the left thorax (*Fig 327*)
- 2 The whole stomach, including both orifices, may be in the thorax



FIG 326—Drawing showing defect in the left diaphragm, due probably to non-development of the left crus. It was closed by peritoneum, which constituted the hernial sac.

The stomach becomes rotated so that the greater curvature is directed cranially (*Fig 328*)

- 3 The stomach may be angulated over the edge of the diaphragm (*see Fig 330*). Again, although a large sac is present, it may sometimes be empty, the



FIG 327—Stomach (1) and portion of colon (2) in the thorax.



FIG 328—The whole stomach is in the thorax, the greater curvature uppermost. 1 Pyloric portion of stomach, 2 Breast shadow.

stomach being in the abdomen. This was proved—unfortunately—in two of my three cases of this type, in one when the X-ray examination was being made, in the other at the first operation.

Each of the three patients with a hernia in this site upon whom I have operated was middle-aged. The symptoms were attacks of severe abdominal pain, often but not always brought on by taking food, so much so that one of the patients said, "I look at food, I want it badly, and I do not take it." Although these attacks were severe, the "bursting" sensation and feelings of oppression were almost worse than the pain. In one there was severe pain in the left shoulder, axilla, and arm, in another severe hæmatemesis had occurred, in all the attacks were becoming more frequent.

Portions of the clinical history of two of the three patients belonging to this type show that—as in an inguinal hernia—the contents may not always occupy the sac. When there are no adhesions and when the orifice is large, as it apparently always is in this type, gravity may tend to keep the sac empty. When symptoms are present and another diagnosis has not established their cause, grave disservice may be done to a patient unless the radiological examination includes inspection in a modified Trendelenburg position. One of these patients had been examined radiologically and sent with a diagnosis of gall-stones, and with the statement that X-ray examination had excluded any abnormality of the alimentary canal. On opening the abdomen, neither stomach, great omentum, nor transverse colon was to be seen. They occupied the thorax, and the aperture of entry was readily found. The barium-filled stomach was certainly in the abdomen and not in the thorax when the X-ray examination was made with the patient in the upright position. At operation the condition was obvious and was dealt with, but in the second case it was not obvious and was not dealt with—at least not until later and by submitting the patient to another operation.

The Sac—In each of the three cases of this type a sac was present—and a very large one, consisting of peritoneum and pleura separated by a little areolar tissue. The pleural envelopment of the peritoneal sac was complete and well-fitting, so that I believe it had been present from an early period of development.

Margins—Antero-laterally there is firm diaphragmatic tissue, but postero-medially when the sac is opened and the margins are examined from the thoracic side there is only pleura and areolar tissue covering the œsophagus, the aorta, and the neighbouring area of vertebra, rib, and intercostal space.

The embryology of the condition in these three patients raises an interesting problem. It is repeatedly stated that in hernia occurring through the pleuro-peritoneal hiatus no sac is present, that therefore it is a false hernia, and that the patients almost never survive. Hume stated in his 1922 Hunterian Lecture that while hernia through the pleuro-peritoneal hiatus is the commonest type—if foetal cases are included—it is rarely met with in adult life, and that Sir Arthur Kieth had only been able to collect 21 cases in the medical museums of London, and of these only 2 survived birth by more than a few weeks. In each of my three cases a sac was present and the patients had reached middle-age. It occurs to me that a review of the embryology may explain this discrepancy. In the early phase of foetal development the pleuro-peritoneal canal is not closed. Any slight lack of co-ordination in development before the closure could lead to displacement of abdominal organs along the open canal—there is no membrane to make a sac—the condition tending to terminate life very soon after birth. This is what is found post mortem in the great majority of infants dying from the condition, and it had led to the widely held belief which has been expressed by so many writers. The

three cases now being described would indicate that in the second period of diaphragmatic development—let us say during the second month—the pleuro-peritoneal canal is in process of being closed by membrane, but is not yet muscularized. This always remains a comparatively weak area. Protrusion of abdominal organs beginning at this stage would carry with it a peritoneal sac and push before it a pleural covering as well. This may take place slowly and may not become extreme until later life, thus enabling the individual to live, although the disability continues to increase, and it explains the presence of the sac and the age at which the condition has been found in my cases.

Etiology—In discussing the etiology of these three cases I want to put forward another explanation which I believe is the right one, but which I cannot yet prove. In the section of this paper dealing with embryology, *Figs 311, 312* showed the central area where muscularization began and its line of spread from the centre to the periphery. The crura are therefore a *final development*, not an *origin* of the muscle. Operation in each of these three cases gave increasing familiarity with the region, and I now suspect that each hernia has resulted from a congenital absence of the left crus and the muscular fibres continuous with it. Certainly in the last two no muscle could be found where the left crus should have been, and no anatomical hiatus existed, a large defect in the diaphragm being present occupied by the sac. Both œsophagus and aorta passed from the thorax to the abdomen behind the sac through the defect. Both these structures were completely visible in their lower thoracic course, and, through the defect in the diaphragm, the junction of the œsophagus with the stomach was under vision. This theory is strongly supported by consideration of the case illustrated by *Figs 333-335* (hernia diaphragmatica transversa), where both crura were undoubtedly absent, the diaphragm having no posterior attachment in the region of the vertebral column. This absence of a postero-medial or posterior margin to the hernial orifice is of importance to the surgeon and to the patient, for there is no firm posterior attachment for the sutures, for this reason, and also because the phrenic nerve has been temporarily paralysed, the repaired diaphragm—and the fundus of the stomach—remains at a level higher than normal (*Fig 329*).

Nevertheless symptomatic cure is obtained, the patients being completely comfortable, and a skiagram taken subsequent to operation shows that the stomach, which had been entirely thoracic and rotated on itself, is now situated in the abdomen with its axis and its function normal.

In the second case of this type the patient had been sent for operation for gall-stones. The history appeared to be characteristic and gall-stones were present. The operation note states that the stomach was abnormally shaped, two pyloric constrictions appeared to be present, one at the usual site and another three or four inches proximal to it, the intervening portion being a cylindrical tube of about duodenal calibre, but with its walls more like stomach than duodenum (*Fig 330*).

Unfortunately, having found the gall-stones, it did not occur to me to examine the diaphragm. The explanation came later. The patient was not cured of the attacks of pain by the cholecystectomy, and a radiological examination subsequently carried out by Dr Lindsay Locke for Dr Gordon Lane revealed the diaphragmatic hernia. In this patient the defect in the diaphragm was very large. The stomach reached the clavicle (*Fig 331*), but as it was completely in the abdomen at the time of the first operation it obviously could slide out of the thorax. The explanation

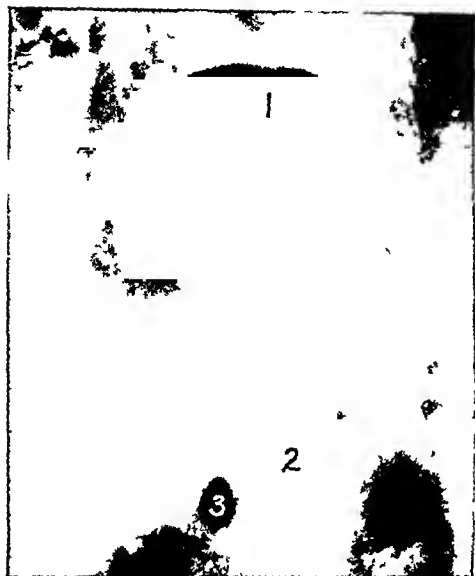


FIG 329—The same patient as Figs 327, 328, after operation 1 Fundus of stomach, 2 Pre pyloric segment, 3 Duodenal cap

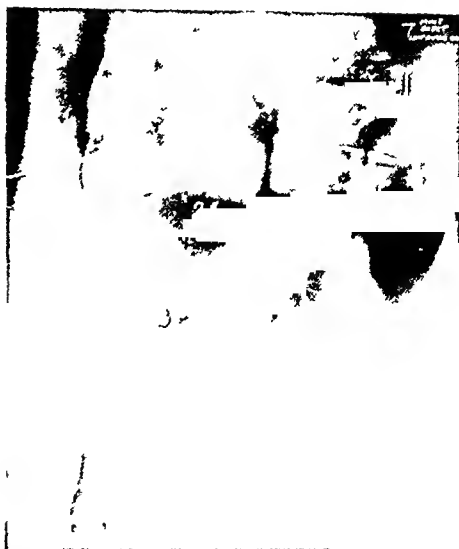


FIG 330—Showing 1 Air cap in herniated stomach, which is seen angulated over the margin of the defect in the diaphragm, 2 Dome of right diaphragm, 3 Abdominal portion of stomach which was permanently narrowed as described in the text



FIG 331—The fundus of the stomach (1) is seen almost reaching the sterno clavicular articulation 2, Colon

of the distal narrowed portion of the stomach is possibly that the proximal portion did not empty easily and became dilated as seen in *Fig 330*, while the distal portion only filled by overflow and remained tonically contracted. This exit by overflow is seen in the duodenum of the patient illustrated in *Fig 338*, where the pylorus was fixed by dense adhesions several inches above the diaphragm.

IV HERNIA IN THE REGION OF THE ŒSOPHAGUS

As stated in the classification, I have examples of hernia in this situation due to three entirely different anatomical defects. *Fig 332* will show at a glance the differences before the more complete description of each is given.

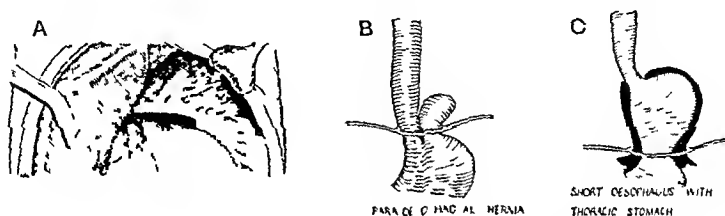


FIG 332—A Congenital absence of both crura and adjoining portion of the diaphragm. The stomach slides up into the posterior mediastinum. B Para-oesophageal hernia. C The œsophagus terminates in the thorax and the cardiac portion of the stomach occupies the thorax.

ŒSOPHAGEAL REGION (A) HERNIA TRANSVERSA DIAPHRAGMATICA

This is due to non-development of the diaphragmatic crura, it is *not* a para-oesophageal hernia occurring through the hiatus. The anatomical hiatus, which should be formed by the fibres continuous with the crura, does not exist. There



FIG 333—Transverse diaphragmatic hernia in which the stomach slides up and, unless held down, occupies a position in the posterior mediastinum as shown in *Fig 334*.

is no posterior attachment of the diaphragm in the region of the aorta, the œsophagus, and the vertebral column. There is a gap in the diaphragm 4 to 5 in in lateral measurement and 2 to 3 in in antero-posterior measurement. This defect is shown in *Fig 333*. This drawing shows the left lobe of the liver

retracted and the stomach withdrawn from the sac which occupied the posterior mediastinum, and represents what was seen at operation. If the hand let go the stomach, the latter at once slipped up through the defect. The concavity of this



FIG 334 —The stomach occupies the sac in the posterior mediastinum

reniform defect is formed by the body of the twelfth dorsal vertebra. The hernial sac protrudes through this into the posterior mediastinum, and the defect is bilaterally symmetrical (*Fig 334*). The œsophagus is of normal length, as shown in *Fig 335*, and at operation its junction with the stomach was clearly defined when the latter organ was—quite easily—drawn down into the abdominal cavity.

Consideration of these two skiagrams suggested the type of hernia. This patient was 54 years of age and she had been under observation for many years.

A short œsophagus had been dia-

gnosed from X-ray examination elsewhere. Our examination (*Fig 335*) disproved this. She had suffered from symptoms for many years and their severity was increasing. Dyspnoea was distressing, and a feeling of tightness and pain was always present across the lower sternal region. Turning in bed caused such distress that she had adopted the plan of sitting up, turning in the desired direction, and then lying down again. The symptoms and the examinations showed that the hernia was increasing in size. This is to be expected, as with every inspiration suction is exerted and, as well, hydrostatic pressure is directed on to the contents of the hernial sac with the constantly recurring expulsive efforts of the body.

At operation, by dividing the left coronary ligament of the liver and retracting the left lobe, the region was completely under vision and could be readily examined. The stomach was easily withdrawn from its sac in the thorax, the reniform orifice of the sac was clearly defined, the aorta and the œsophagus were visible and palpable behind the peritoneum of the sac.



FIG 335 —œsophagus of normal length. The stomach is in the posterior mediastinum

This type would seem to be due to the absence of development of both crura and the adjoining part of the diaphragm, leaving a gap between a well-defined and

perfectly free posterior margin of the diaphragm which bounds the sac anteriorly and the vertebral column and adjoining part of the paravertebral grooves which bound it posteriorly. Through this gap the peritoneal sac ascends into the posterior mediastinum. The defect is therefore behind the incomplete diaphragm. In closing the gap by sutures a firm grip can be obtained anteriorly, but, as in the last type, there is no strong structure posteriorly into which the sutures may be inserted. In spite of this, closure of the gap has given complete relief to the very distressing symptoms.

In this patient gall-stones were present. The operation had been an extensive one, and I considered it expedient to leave them to be dealt with at a later date, if necessary.

In another patient, whose symptoms pointed to gall-stones, the X-ray examination (*Fig 336*) revealed a diaphragmatic hernia as well as a pathological

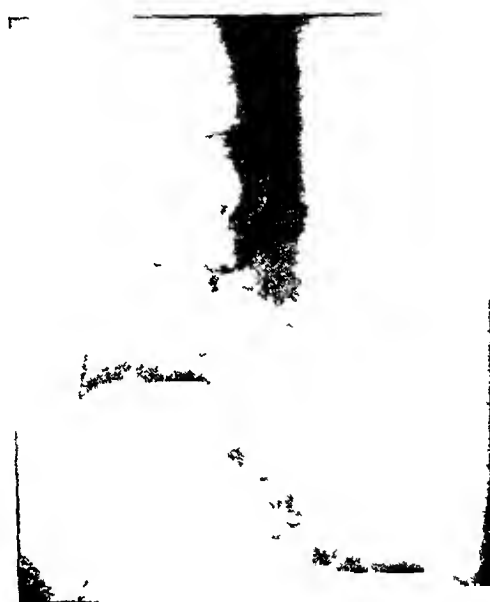


FIG 336 —The upper loculus is in the thorax.

gall-bladder. Operation for the gall-stones gave the opportunity of examining the hernial orifice, and it proved to be similar to the one just described. As in this case the symptoms were due to the gall-stones alone, they were dealt with and the hernia left. The patient's husband is a medical practitioner and she can be kept under observation.

I have seen a third patient whose hernia I believe to be of this type, absence of the crura leaving a gap between the posterior margin of the diaphragm and the vertebral column, which allows the stomach to slide up into the thorax carrying the lower end of the œsophagus up with it (*see Fig 349*). This patient suffers from attacks of intense oppression and dyspnoea which last for a varying number of hours. These attacks are separated by several months of freedom. The condition in this patient has not been confirmed by further X-ray examination nor by operation.

ŒSOPHAGEAL REGION (B) PARA-ŒSOPHAGEAL HERNIA

The second type of hernia in the œsophageal region is the para-œsophageal. The œsophagus is of normal length. The hiatus is present, but it becomes dilated. A hernial sac exists, or is acquired, passing up through the hiatus, lying beside the œsophagus.

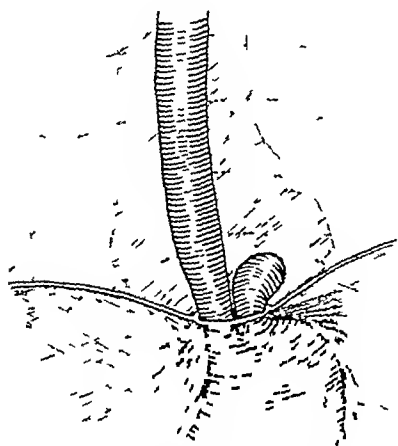


FIG 337—Initial stage in formation of para-œsophageal hernia

Fig 337 illustrates the condition. This type has been well described, but each of my two cases has some special features. In the first the symptoms became urgent at the age of 77 and were so severe that the patient retched and vomited constantly and could not retain even arrowroot. Operation was therefore a matter of urgency and was carried out July 2, 1928. The hernia was approached by the abdominal route. This lady is now 82 years of age and, six years after operation, enjoys life—and her cigarettes—very thoroughly.

The second patient, aged 50, had never been happy for over twenty years. She suffered from attacks of pain "like acute indigestion", often with sickness. Diet restrictions had to be made more rigid in recent years, she could never go out to dinner but latterly discomfort was never absent. The appendix had been removed. Subsequently gall-stones were found present and were removed. X-ray examination from time to time showed an increasing amount of the stomach in the thorax. The pylorus was evidently fixed well above the level of the diaphragm and its outlet so hampered that stomach contents only left it in a trickle (*Fig 338*), the stomach itself becoming increasingly distended.

OPERATION—The abdomen was opened through a left subcostal incision. Adhesions, fixing the small portion of the stomach still remaining in the abdomen, were so dense that it was impossible to withdraw the organ from the thorax. A thoracic approach was therefore made. The sac was adherent to the pericardium and to the base of the lung. After freeing the sac sufficiently to open it safely, the stomach was found folded on itself, the greater curvature uppermost, and adherent almost all around the neck of the sac. The adhesions were especially dense in the pyloric region, which was fixed several inches

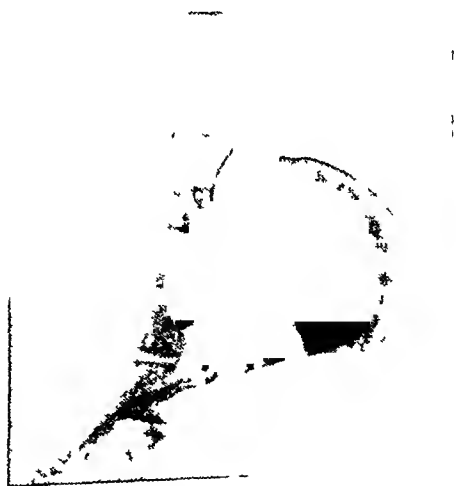


FIG 338—Stomach with greater curvature uppermost, pylorus fixed above diaphragm, barium leaving pylorus in a thin trickle

above the diaphragm. All these features are seen in the skiagrams (*Fig 338*). Separation of the adhesions was continued from the abdominal side, but it was still very difficult to free the pylorus, which remained firmly anchored above the diaphragm, and until this was accomplished it could not even be seen how the stomach should lie. When the adhesions were completely divided the pylorus at once fell into its natural position and—except for its dilatation—the stomach appeared normal. The operation had been long and difficult, and it was essential to close the sac and the diaphragm from above in the speediest manner. The skiagrams showed, and the operation findings confirmed, that this stomach could not pass on its contents except in a small trickle. It could only be dealt with by a double approach.

ŒSOPHAGEAL REGION (c) THE SHORT ŒSOPHAGUS TYPE OF HERNIA

The third type of diaphragmatic hernia occurring in the œsophageal region is that associated with a congenitally short œsophagus, the cardia and portion of the stomach being situated within the thorax. Some have dismissed this as being a simple extension of the gastric mucosa through the cardia into the lower end of the œsophagus, and of no consequence. Gastric mucosa may extend through the cardia, but that is not the condition present in 14 patients whom I have seen in the last few years, and described by Bailey,⁸ Akerlund, Woodburn, Morison, Hurst, Monkhouse, Montgomery, and others. It is astonishing how frequent this condition is. It is definitely the cause of symptoms, not infrequently distressing and sometimes fatal. The embryology has been described by Keith. The stomach originally is almost cervical. Normally the œsophagus elongates and the stomach migrates tailwards until it reaches its position below the diaphragm. In the cases under discussion the œsophagus failed to elongate adequately, and the stomach is partially arrested in the posterior mediastinum (*Fig 339*). This arrested portion of the stomach is surrounded—except at the actual cardia—by a peritoneal sac, so that a true hernia results.

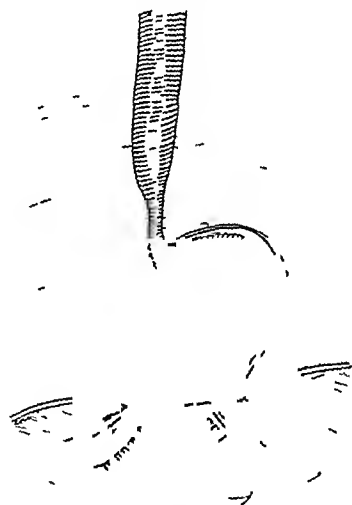


FIG 339—Congenital short œsophagus early stage

If the condition remained at this stage, possibly little harm would result, and indeed it must exist in a number of people who never suspect it, but as it has only been discovered in the patients of myself and others because a reason for some disability was being looked for, we must believe that sooner or later it begins to give trouble, and there is ample evidence that the trouble is progressive.

Anatomy.—That it should be progressive is obvious when we consider the anatomy. The diaphragm should enclose the narrow cardia within its hiatus, but it encloses instead the circumference of the fundus of the stomach by means of a large opening. The stomach is therefore hour-glass-shaped, the cardiac loculus being within the thorax and the pyloric loculus within the abdomen. By radio-scopic barium emulsion is seen to flow readily from one sac into the other.

(Fig 340) Two forces act on this continuously Every expulsive effort of the body is exercising hydrostatic pressure on the stomach and its contents, pushing it up, and during every inspiration it is being drawn into the thorax by suction



FIG 340—1 Cardia, 2 Proximal loculus of stomach in the thorax, 3, Level of diaphragm
4 Distal loculus of stomach in the abdomen A larger thoracic loculus is shown in Fig 342



FIG 341—1 Oesophagus, 2 Cardia, 3 Thoracic loculus, 4 Dome of left diaphragm

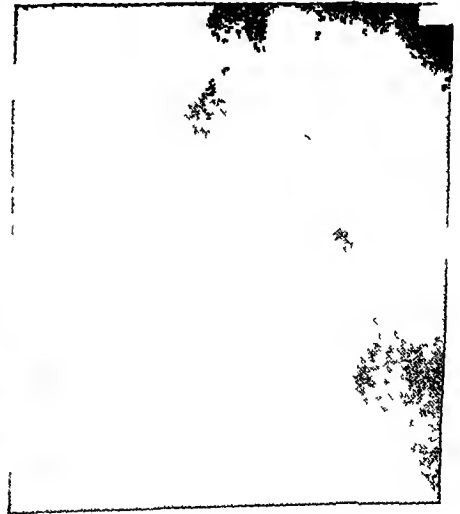


FIG 342—A large thoracic loculus and rotation beginning

The diaphragmatic opening becomes more dilated, and progressively more of the stomach comes to occupy the thorax As this happens, the stomach tends to rotate on its own axis, probably because the greater curvature is longer and more free than the lesser curvature, and—through fixation by adhesions—it may also

become twisted on its long axis. Skiagrams of three patients (*Figs 341-343*) show the successive stages of this passage into the thorax and rotation.

Fig 341 shows an early stage with the thoracic segment small and the axis of the œsophagus and the stomach normal. *Fig 342* shows a later stage—the thoracic segment of the stomach is increased at the expense of the abdominal segment and rotation is beginning. *Fig 343* shows a very late stage. The drawing (*Fig 344*) explains the film, which was given to me by Dr A F Hurst. The

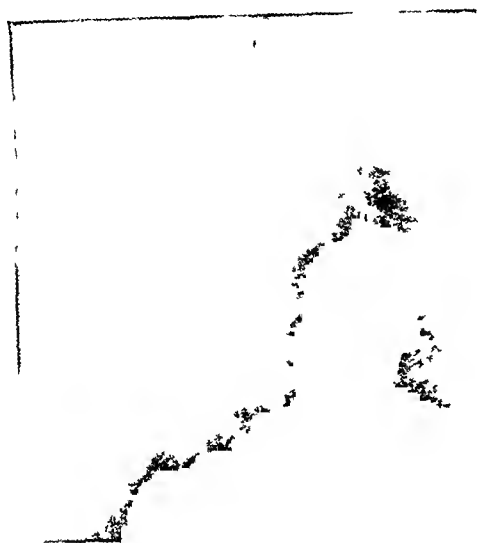


FIG 343—Practically the whole of the stomach is in the thorax, and rotation is almost complete

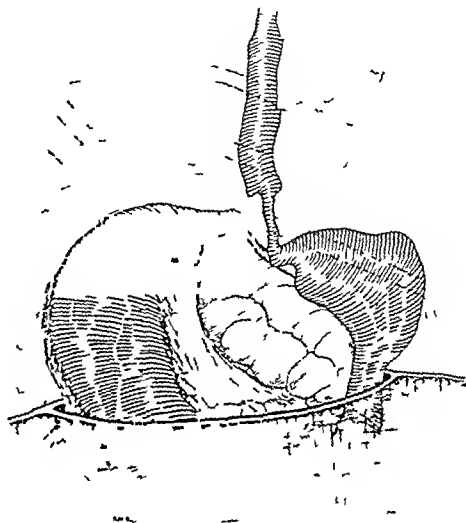


FIG 344—Drawing from film to explain *Fig 343*

patient was very ill and the skiagram was taken under difficulties, but it shows the short œsophagus and the stomach in the thorax with its greater curvature uppermost. Some bowel has been drawn or forced up into the thorax. The stomach is twisted or obstructed in its middle third, resulting in two loculi. Emptying the proximal loculus by an œsophageal tube gave relief. It was impossible to empty the distal loculus.

It would not add to the value of this article to include skiagrams of the other patients, but I have films of all of them, and some are used to illustrate pathological changes incidental to the condition.

Pathology—Apart from increasing herniation, pathological changes may, and do, occur. (1) At the cardia, (2) In the œsophagus, (3) In the stomach.

As the cardia is situated well above the diaphragm in the thorax it is very accessible for X-ray examination. The first patient seen with this condition came in 1930 bringing a skiagram (*Fig 345*). From this a diagnosis of carcinoma of the œsophagus had been made. I knew he had suffered from dysphagia for five years previous to this, and therefore felt sceptical about the diagnosis. X-ray and œsophagoscopic examinations had been made over this period without result. I then had the examination carried out in the Trendelenburg position, when the condition was obvious (*see Fig 340*). This patient has been watched for four more years—nine in all. Dysphagia has been at times extreme with choking and return

of food Is the dysphagia due to spasm (cardiospasm or achalasia), or is it due to organic stricture?

Dilatation—That cardiospasm or achalasia occurs at the cardia (situated in the thorax) with dilatation of the œsophagus is shown by *Fig 347*

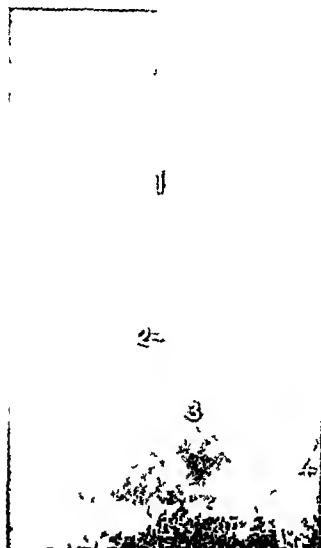


FIG 345—1 Dilated œsophagus, 2 Cardia, 3 Thoracic stomach (1930)



FIG 346—The same patient as *Fig 345* in 1933 Each dome of the diaphragm is well seen with portion of the stomach above



FIG 347—Dilated œsophagus Cardia in the thorax Portion of stomach in the thorax Both domes of the diaphragm are seen

Stricture—The clinical, radiological, and pathological evidence indicates that the stricture may be organic. Clinical, because a small mercury bougie would pass in the beginning and a larger one would not—now I cannot pass a mercury bougie at all, nor can I in several other patients. The X-ray evidence is seen in *Fig 348*, a skiagram which shows the dilatation above—not to an extent sometimes seen in cardiospasm certainly—and in addition we see an irregularity in the œsophagus immediately above the constriction which œsophagoscopic examination shows to be due to ulceration. Pathological evidence has been obtained from actual inspection and palpation at operation. This confirmed that organic constriction was present. One patient had been admitted as a case of œsophageal obstruction. She improved and was discharged. She was later re-admitted in



FIG 348—Showing dilated œsophagus, a narrow stricture at the cardia with an ulcer above the narrowing, the cardiac portion of the stomach in the thorax.



FIG 349—The œsophagus is seen entering a large thoracic loculus of stomach. The diaphragm is clearly seen, as is the pyloric portion of the stomach and the duodenum.

a condition of acute obstruction, thought to be too ill for X-ray examination, and a gastrostomy was performed. While this was being carried out by a colleague the stomach and diaphragm were examined, and the hernia with its peritoneal sac was discovered. X-ray examination revealed the true diagnosis. I had hoped to bring the stomach up to the œsophagus above the stricture, after paralysing the left phrenic nerve. This was impossible, but through a thoracic approach I dissected the stenosed area cleanly and found it to be about 3 cm long, fusiform, and very firm.

One patient with short œsophagus has never, up to the age of 27, been able to swallow anything firmer than mince. Attempts to pass a mercury bougie have failed. He has to give his whole attention to his meal, and takes a long time over it. He can never go out to meals, and he is undersized. I have recently seen a child of 12 who has never been able to swallow properly. In this child the results of malnutrition are obvious.

Dysphagia is not due solely to spasm or stenosis. Remembering the anatomy

and the pathological physiology it will be understood that the thoracic loculus of the stomach will be sometimes distended. If the stomach is angulated across the diaphragmatic opening, or even partially rotated, the distension of the thoracic sac will be increased, maybe very greatly, and it may press on or kink the lower end of the œsophagus, causing dysphagia, or on the pericardium, causing oppression and dyspnoea. I regard this as the reason why the dysphagia is sometimes so strangely inconstant. It may appear to be complete in recurrent attacks, and later the patient may swallow mince and soft foods readily. I have also known the attacks of dyspnoea and of what the patient calls "unendurable oppression" occur only once or twice a year, the radiogram showing a large loculus of stomach in the thorax (*Fig 349*).

Ulceration—This has been shown in the œsophagus immediately above the stricture (*see Fig 348*). It is associated with constant pain around the left costal margin. I have not had a case of gastric ulceration in this condition, but Hurst and his co-workers have published a detailed account of two in which the clinical records—including severe hæmatemesis—the radiological evidence, and the post-mortem findings, were complete. Two of the patients with short œsophagus and severe dysphagia have been under the care of Mr V E Negus also. In one of these the œsophagus was filled with fluid, as may occur in achalasia. In each of them endoscopy revealed the presence of some ulceration just above the narrowing, and in each it was possible to pass a dilating bag through the narrowing under vision and dilate it.

In conclusion, I have operated upon 8 patients with diaphragmatic hernia in whom the œsophagus was of normal length, 6 of these are cured, one had complete relief of symptoms but died of a pulmonary embolus ten days later. In the last one operated upon recently pneumonia occurred in the lower lobe of the opposite side—that is, the side not operated upon. This did not resolve, and a little over four weeks after the operation the patient died. A post-mortem examination was permitted. The left lung—the side from which the rib had been excised—had expanded. The diaphragm had healed and the line of suture where the hernia had been closed could not easily be found. The œsophagus entered the abdomen normally, and the stomach, which had been entirely in the thorax, occupied its normal abdominal position. The operation had achieved cure of the hernia and restoration of the stomach to the abdominal cavity. Pneumonia has, naturally, to be regarded as a possible complication of the operation. Patients with short œsophagus and partial thoracic stomach suffer in diverse ways, and treatment must in the main be symptomatic. Some only require occasional advice. In some the passage of a mercury bougie is sufficient. In some a dilating bag should be passed under vision.

Experimental work recently carried out by G C Knight,⁹ late House Surgeon to the Surgical Professorial Unit, St Bartholomew's Hospital, at the Buckston Browne Research Farm of the Royal College of Surgeons, has given hope that excision of the sympathetic plexus surrounding the cœliac axis may relieve the cardiospasm (or achalasia) where this has not already resulted in organic stricture.

In diaphragmatic hernia the symptoms of the condition simulate those due to gall-stones, and it is interesting that in the 11 patients with œsophagus of

normal length and herniation of the stomach, gall-stones have been found present in 5

It should not be assumed that operation can be carried out in cases such as these without risk, or that convalescence is always smooth. The difficulties of the operation vary greatly with the situation of the defect, and with its accessibility, and this will naturally affect the convalescence.

SUMMARY

- 1 Cases of diaphragmatic hernia are less rare than is generally recognized
- 2 The type associated with short œsophagus is by no means uncommon
- 3 The symptoms may be very distressing and are often grave
- 4 The symptoms are such that they strongly suggest their origin
- 5 X-ray examination can do much more than determine the presence of a hernia, by careful posture of the patient and a planned technique the type and site can be determined
- 6 In cases with an œsophagus of normal length an operation can be performed that frees the patient from danger and completely relieves the symptoms. The operation is not without some risk. This is not greater than one would expect, while without operation the symptoms are very distressing and the risk of a fatal termination is probably more frequent than when an operation is performed
- 7 In cases with short œsophagus and thoracic stomach a radical operation to cure the condition has not yet been carried out, but much can be done to ameliorate the condition

I am indebted to the following radiologists for the excellent skiagrams which illustrate this article: Drs Lindsay Locke, Stone, Finzi, Simon, Bull, Coldwell, and Shanks

REFERENCES

- ¹ KEITH, Sir A, *Brit Med Jour*, 1914, II, 1927
- ² HUME, J B, *Brit Jour Surg*, 1922, X, 207, 1932, XI, 527
- ³ MORISON, J M, *Proc Roy Soc Med*, 1930, XXIII, Sept
- ⁴ HURST, A F, COLLIER, W, and SHEAF, E W, *Guy's Hosp Rep* 1929, April, 159
- ⁵ HEDBLUM, C A, *Jour Amer Med Assoc*, 1925, Sept 26
- ⁶ HARRINGTON, S W, *Jour of Thoracic Surg*, 1931, I, Oct, No 1
- ⁷ BARRETT, N R, and WHEATON, C E W, *Brit Jour Surg*, 1934, Jan, 420
- ⁸ BAILEY, P, *Anat Record*, 1919, XVII, 107
- ⁹ KNIGHT, G C, *Brit Jour Surg*, 1934, July, 155

CONGENITAL HERNIA THROUGH RIGHT DOME OF DIAPHRAGM

BY FRANK FORTY

RESIDENT SURGICAL OFFICER THE GENERAL HOSPITAL, BIRMINGHAM

ALTHOUGH diaphragmatic hernia is not a particularly rare condition, several features in the case about to be described appear to justify recording it (1) The hernia, almost certainly congenital in origin, was the cause of acute intestinal obstruction in a man aged 70 years, who had never previously suffered from his defect (2) The hernia occurred in the right dome of the diaphragm This situation is stated to be the least common of the various sites at which herniation may occur It is also the type of hernia which is the most difficult to explain on embryological grounds (Hume¹)

D C, male, aged 70, was admitted to the General Hospital, Birmingham, under Mr J B Leather, on Nov 8, 1933

HISTORY —The patient complained that his bowels, previously regular, had not been opened for the past six days During this period he had suffered from general abdominal pain, had vomited frequently, and had been hiccuping during the last few days He had previously been perfectly healthy and had never been troubled by indigestion or constipation Many years ago he had had a kick in the chest when he had fallen from a horse

ON EXAMINATION —The patient seemed to be in good general health The abdomen was markedly distended, especially centrally and in the epigastrium There was general tenderness Free fluid was detected

OPERATION —Under general anaesthesia I opened the abdomen by a sub-umbilical right paramedian incision Blood-stained free fluid was present The whole of the small intestine was distended and congested The caecum was not present in the expected position in the right iliac fossa, and on searching for it towards the hepatic flexure, a circular opening was felt in the right dome of the diaphragm Through the opening about the last 3 ft of small intestine had passed into the right pleural cavity, and the constriction due to the margins of the diaphragmatic opening was the cause of the intestinal obstruction The caecum was drawn up immediately below the diaphragm, and the appendix had accompanied the small intestine through the opening

The hernia was reduced without difficulty, and the affected loop of intestine was found to be viable On withdrawing the intestine from the hernia, the right lobe of the liver, which had been dislocated to the left, returned to its normal position, thus guarding the opening in the diaphragm The abdomen was closed

The patient died, apparently from paralytic ileus, a few hours after the operation

POST-MORTEM EXAMINATION —

Thoracic Viscera —These were normal

CONGENITAL DIAPHRAGMATIC HERNIA

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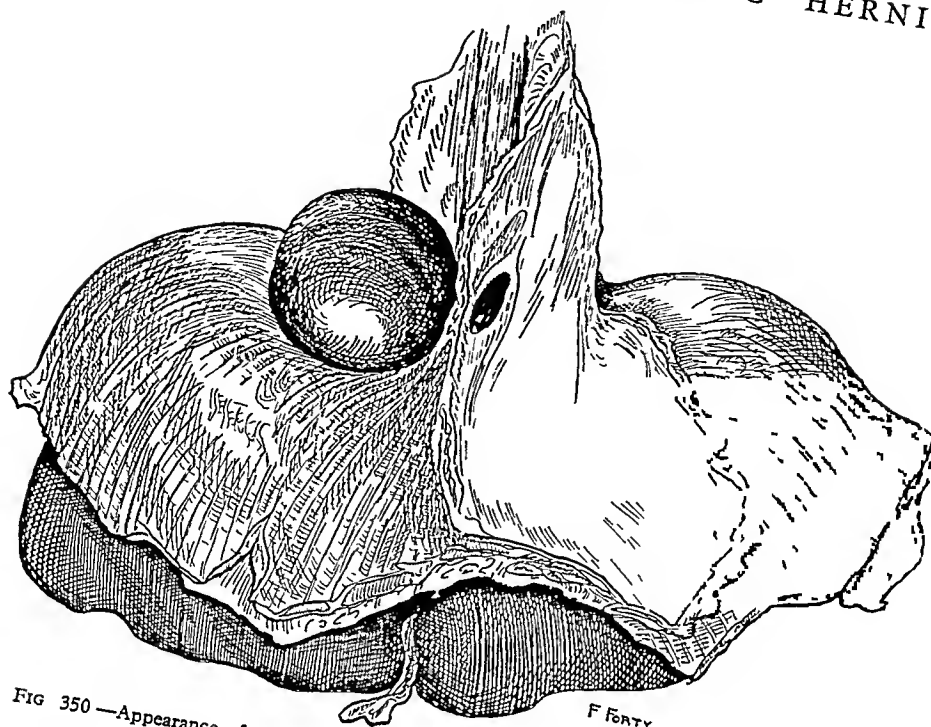


FIG 350 —Appearance of the upper surface of the right lobe of the liver herniated into the right pleural cavity

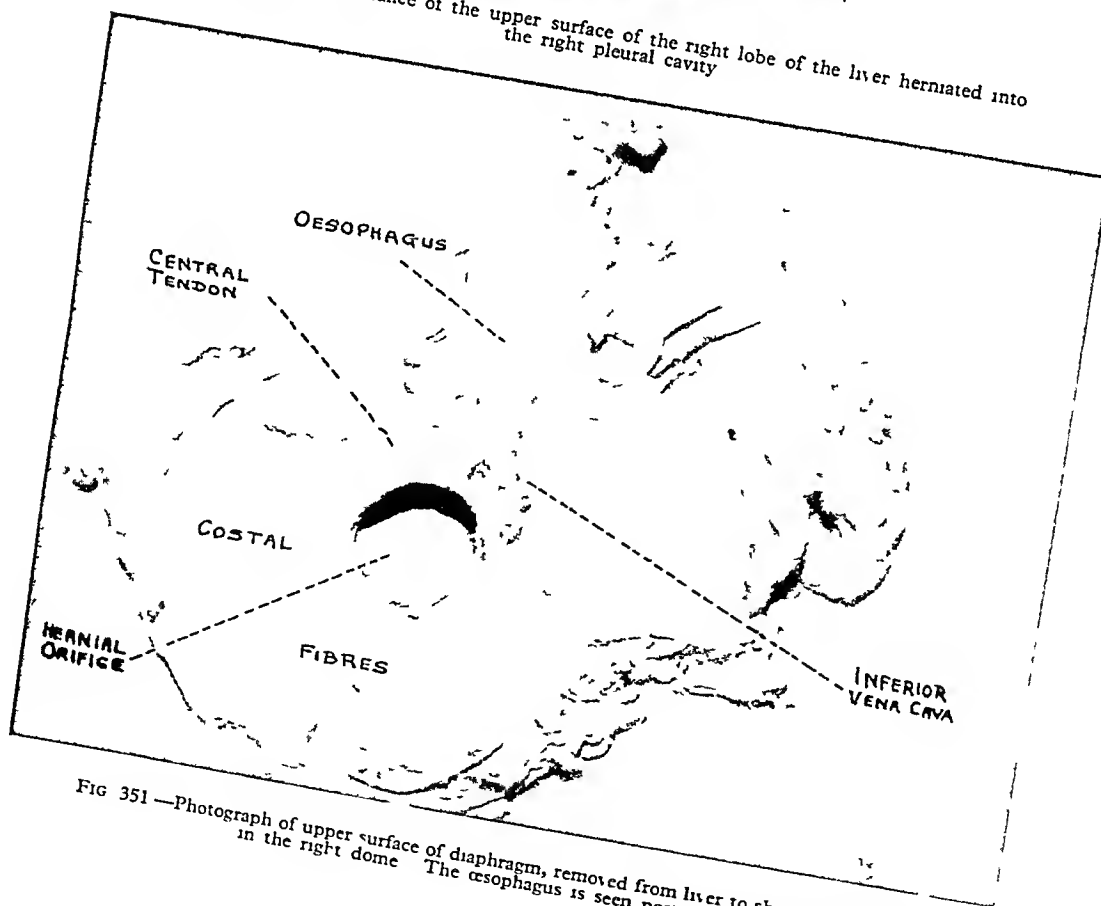


FIG 351 —Photograph of upper surface of diaphragm, removed from liver to show the opening in the right dome The oesophagus is seen posteriorly

Abdomen—The small intestine was distended throughout, and the constriction groove from the strangulation was clearly seen. The cæcum was very mobile, and now occupied the right iliac fossa. The other abdominal viscera were normal, except that a portion of the upper surface of the right lobe of the liver was herniated through the opening in the diaphragm, which it completely filled (*Fig 350*).

Diaphragm—An almost circular opening, $2\frac{1}{2}$ in diameter, was present in the right dome (*Fig 351*). It was situated immediately to the right of the pericardium. It was bounded posteriorly by the right leaf of the central tendon, and anteriorly and laterally the costal muscle fibres of the diaphragm ended in its margin. Through the opening the pleural and peritoneal cavities communicated freely. No hernial sac was present. The pleural and peritoneal serous membranes were continuous over the margin of the opening, which was perfectly smooth, and free from adhesions.

DISCUSSION

Hume¹ classifies diaphragmatic herniæ into (1) Congenital, (2) Acquired—(a) non-traumatic, (b) traumatic.

Our knowledge of congenital diaphragmatic herniæ may be briefly summarized by the statement that they most frequently occur at the site of the embryonic pleuroperitoneal canals, where there is a failure of fusion between the costal and vertebral (crural) sets of muscle fibres (*Fig 352*). Such a hernia is more frequently

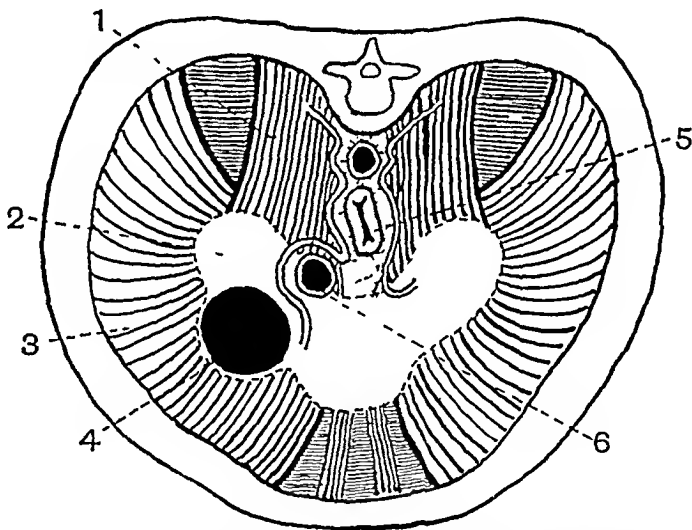


FIG 352.—Diagram of upper aspect of diaphragm to show relation of opening to the central tendon, costal muscle fibres, and pericardium. 1 Vertebral fibres, 2 Central tendon, 3 Costal fibres, 4, Hernial orifice, 5 Œsophagus, 6 Inferior vena cava.

seen on the left side, perhaps because the diaphragm is not here supported by the liver to the same extent as it is on the right. Occasionally, congenital herniæ occur through other openings, such as that for the œsophagus, and complete absence of the left half of the diaphragm occurs in a number of cases. Hernia through the right leaf of the central tendon, on the other hand, seems to be particularly rare.

Keith² described 34 specimens of diaphragmatic herniæ which he found in various London museums, and of these only 2 were through the right dome. One had a sac, and the other, occurring in a child, had no sac, the pleural and peritoneal cavities communicating freely through the opening, which was occupied by a process of liver projecting into the right pleura. This case appears to correspond exactly to the one recorded in this communication. In his description of these two cases Keith states that there is little doubt that they are congenital in origin and that they were produced by an abnormal development of the liver within the septum transversum of the embryo.

Hume,¹ discussing those herniæ which occur at the site of insertion of the muscle fibres of the diaphragm into the central tendon, suggests that these may arise from a sudden forceful contraction of the diaphragm resulting in a rupture of the muscle fibres from their insertion. He accordingly infers that these herniæ through the dome of the diaphragm which are provided with a sac belong to his "acquired non-traumatic" group, but, in agreement with the opinion of Keith quoted above, he believes that herniæ occurring in this situation without the presence of a sac are congenital in origin. Hume reserves his "acquired traumatic" group for herniæ resulting from direct penetrating wounds of the diaphragm, such as those caused by the ends of fractured ribs, and emphasizes that adhesions are almost always present at the margins of such an opening.

CONCLUSION

In the light of the available evidence concerning the formation of diaphragmatic herniæ, the case here recorded appears to be one of congenital origin occurring through the right dome.

I am indebted to Mr J B Leather for permission to record this case, and to Professor Lockhart for his assistance and advice.

REFERENCES

- ¹ HUME, J B, *Brit Jour Surg*, 1932, vii, 527
- ² KEITH, ARTHUR, *Brit Med Jour*, 1910, ii, 1297

INTESTINAL HERNIATION THROUGH A MESENTERIC HIATUS

By E S J KING,

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FROM time to time many peculiar forms of intestinal obstruction have been described. Some of these are but anatomical or pathological vagaries. Others, though very rare, show features of special interest, and one of these is here described. Several years ago I saw the following case —

Case 1—A female, aged 56 years, complained of abdominal pain of a colicky type, umbilical in position, associated with vomiting and complete constipation, which had been present for three days. For six hours the pain had been more severe. She had had no previous illnesses of note. On examination there was generalized distension, showing a 'ladder pattern'. Her general condition was fairly good. Enemata gave no result.

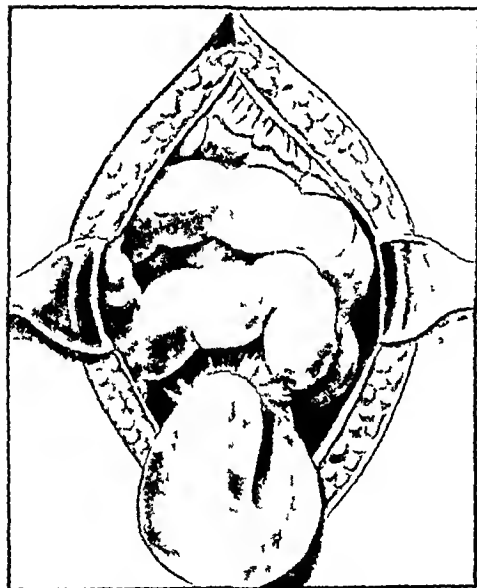


FIG 353—*Case 1*. The appearances found at operation (first case) showing herniation of the second last loop of ileum through a hiatus in the mesentery of the last loop.

Laparotomy was performed and a herniation of the second last loop of ileum through the mesentery of the last loop was found (*Fig 353*). The strangulated loop was markedly distended and the wall was bluish. The hiatus in the mesentery had a definite free fibrous margin in half of its extent, in the remainder the bowel was adherent to the ring. The mesentery through which the gut passed was damaged during reduction of the loop, and excision of both loops was performed. An anastomosis between the ileum and the cæcum (anterior tænea) was made. The patient made an uninterrupted recovery.

The formation of such hiatuses in the mesentery seemed difficult of explanation. The usual hypotheses are that they may be (1) Due to operation, an opening in the mesentery being incompletely closed, (2) Due to injuries which rupture the mesentery, and (3) Congenital defects. This patient had not had any operation performed and gave no history of abdominal injury. The third suggestion, in the absence of any direct evidence from foetal study, affords me, personally, no satisfaction.

Frequently the causes of conditions are divided into 'congenital' and 'acquired' varieties. This use of these terms is wrong in so far as it necessitates an attempted comparison between factors of two different kinds. The term 'congenital'

presupposes that the condition is present at birth, and 'acquired' indicates that the causal factor is extraneous in origin. Thus an attempt is made to compare a time-factor with a true etiological factor. Gradually, no doubt in order to overcome this difficulty, the term 'congenital' has been used in the etiological sense and to mean an inherited factor. Such usage can only be misleading.

The etiology of groups of conditions should, then, be divided into 'hereditary' and 'acquired', according to whether the etiological disturbance is intrinsic in or extrinsic to the tissues. Acquired conditions may occur in either antenatal or postnatal life, and if the former are referred to as being 'congenital'—and of course they are congenital in the time sense of the term—it must be appreciated

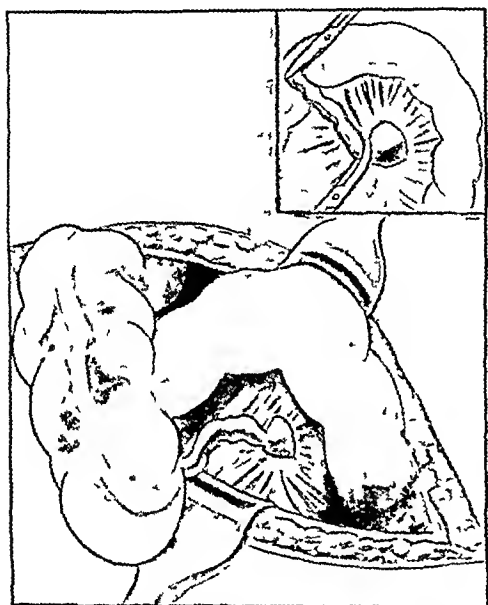


FIG 354—Case 2. The appearances seen at operation. The layers of the abdominal wall are not shown. The appendix is seen lying attached to the inferior aspect of the mesentery of the last loop of ileum. At the tip of the appendix is a bulbous structure. *Inset*. The appendix being removed by retrograde method and a forceps applied to bulbous area.

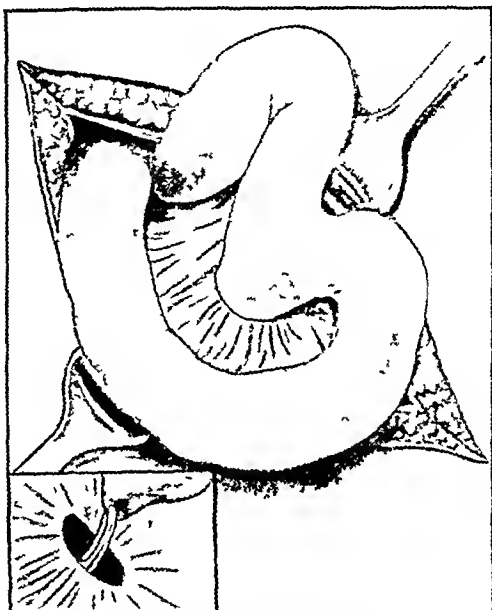


FIG 355—Case 2. The cæcum returned to the abdominal cavity and the last loop of ileum turned over (cf Fig 354) so that the mesentery of the last loop of ileum can be viewed from above. The second last loop of ileum is seen to be attached to the mesentery. *Inset*. Traction resulted in the bulbous structure (Fig 354), to which an artery forceps was attached, being drawn through the mesentery.

that the term only indicates the period at which they occur and does not give any indication of the cause of the disease.

For these reasons the 'congenital' origin of the hiatus in the mesentery was, at least temporarily, dismissed.

At intervals, this case was given much, albeit fruitless, consideration. Recently, however, observations on another case suggested possible explanations of earlier findings.

Case 2—A female, aged 30, was admitted to hospital suffering from an attack of acute appendicitis. She had had pain in the right iliac fossa for three months prior to admission.

At operation the appendix was found to be inflamed and attached to the inferior aspect of the mesentery of the last loop of ileum (Fig 354). Owing to difficulty in separation it was removed by the retrograde method (Fig 354, inset). When almost

completely freed the tip was found to be markedly bulbous and strongly attached to the mesentery

Further dissection showed that the bulbous portion passed through a hiatus in the mesentery. At this stage the appendix suddenly separated at the junction with the bulbous portion, which had a curiously vascular soft wall. A forceps was placed on the bulbous portion. Examination of the appendix showed that the end was not open but was apparently the true tip of the organ and that the bulbous part was extraneous.

The last loop of ileum was now turned over (*Fig 355*) and the second last loop was found at one point to project through the hiatus in the mesentery of the last loop. When traction was exerted, the invaginated portion and the attached forceps were pulled through (*Fig 355*, inset) from the far side. A small opening in the bowel was over-sewn. It was noticed that the mesentery in the region of the hiatus was curiously avascular and was thinner though somewhat tougher than usual.

The patient made an uninterrupted recovery.

The sequence of events in this case appears to be as follows. An attack of appendicitis resulted in the attachment of the organ to the ileal mesentery. As the result of the spread of inflammation from this, obliteration of the blood-vessels and fibrosis resulted in avascularity of the area and the formation of a tough though thin area. Spread of the inflammatory process through the mesentery gave rise to attachment of the second last loop of ileum to the mesentery, and with subsequent fibrosis the bowel became drawn into apposition with the affected mesenteric area and then into juxtaposition with the tip of the appendix. The disappearance by atrophy of a small part of the mesentery then allowed of the passage of the piece of bowel. Of course alternatively the mesentery may have been destroyed during the acute stage of the inflammation, but this is immaterial from the point of view of the present thesis.

Two well-known and well-recognized phenomena allow of the correlation of this case with the former. (1) Once the beginnings of a hernia are present, this will readily increase in size, and (2) Adhesions and evidences of inflammation disappear from the peritoneum in a remarkable manner, often without leaving any apparent trace.

It does not necessarily follow that all cases of the formation of an opening in the mesentery are due to appendicitis. Other inflammations may also give rise to the same result. It is noteworthy that most, if not all, of these mesenteric openings are found in the mesentery of the lower part of the ileum. This is the site of election for many inflammatory conditions of the bowel—tuberculosis, typhoid fever, as well as many non-specific conditions.

The essential conclusion is that openings in the mesentery through which bowel may herniate may be the result of inflammatory damage to the mesentery—thrombosis of vessels, fibrosis, and atrophy* of part of the tissue—and the possibilities of such an origin should be thoroughly explored before a slit in the mesentery is regarded as an hereditary defect.

* Since writing the above I observe that Barnard (*Contributions to Abdominal Surgery*, 1910, 207) includes atrophy as one of the causes of slits in the mesentery.

DYSCHONDROPLASIA (OLLIER'S DISEASE) : WITH REPORT OF A CASE

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INTRODUCTION

THE disease which forms the subject of this communication has been given a variety of names. The title here chosen is that given by Ollier (1899), who appears to have been the first to recognize it. Recently there has been a tendency to regard all congenital lesions of the growing ends of bone as being manifestations of the same primary defect. There is, however, only the most superficial evidence that this is the case, and such a view seems only to have arisen because knowledge of these disorders is largely limited to that obtained by radiographic methods. In particular, hereditary deforming chondrodysplasia, diaphysial aclasis or hereditary multiple exostoses, and Ollier's dyschondroplasia are often regarded as the same disease (Coon, 1911, Ashhurst, 1916, Ehrenfried, 1917, Voorhoeve, 1924), but the grounds for this claim are nowhere clearly stated. The view seems to have arisen because in both conditions there may be abnormal areas of cartilage, exostoses, and broadening of the metaphyses, but it only requires a careful examination of radiograms to distinguish a number of points of difference. In diaphysial aclasis hereditary transmission is a very striking feature. In Ollier's disease there is little if any evidence of this. Until knowledge is further advanced, it is wiser to maintain the distinction between these two conditions and not refer indiscriminately to the whole group as the chondrodysplasias. Admittedly there is a number of cases (Voorhoeve, 1924, Dahl, 1930) which is intermediate and cannot at present be placed in any category.

By 'Ollier's dyschondroplasia' we understand a disease of the growing ends of bone in which the normal ossification of cartilage fails to take place, so that, as the bone increases in length, there remain in the diaphyses areas of cartilage which do not ossify. The process is not primarily one of tumour formation, although in some cases, particularly in the long bones of the hands and feet, there may be a hyperplasia of cartilage. That the pale areas shown in radiograms are really cartilage has been amply confirmed by histological examination both in our own case and those of Kohler (1905), S. Johansson (1916), C. Johannessen (1923), Cole (1926), Cleveland (1928), and Sacerdote (1931).

Though the disease shows a tendency to be unilateral, the case which Ollier originally described was not strictly unilateral, since both hands were affected. Some cases have had only one bone or one limb affected, some have been unilateral, some almost entirely unilateral, and some bilateral. They have all had a similar clinical course and pathological findings. It has been found impracticable

to make a complete list of the bilateral cases because they have been published under widely different names. The search would cover some 4000 records of cases of congenital bone 'tumours'. There is appended, however, a list of 29 unilateral and 10 almost entirely unilateral cases which can be accepted as dyschondroplasia. There is reason to believe that the bilateral cases would not exceed this number. Except where otherwise stated in the bibliography reference has been made to the original articles. Such reference has led us to omit as irrelevant several cases appearing in the lists set out by other authors.

PUBLISHED CASES OF OLLIER'S DISEASE

<i>A—Distribution Strictly Unilateral</i>	<i>B—Distribution Almost Entirely Unilateral</i>
Adams	Jansen, J W F
Anschütz	Jansen, M (2 cases)
Bentzon	Johannessen, C
Boinet and Stephen	Johansson, S
Bojesen	Kohler
Brockman	Kummer
Bromer and Rutherford	Lindstrom
Burchard (2 cases)	Richard and Dupuis
Cleveland	Sceerdote and Bonomini
Cole	Thomson
Flotow (2 cases)	Valentin
Hessenthaler	Weber
Huber and Advemer	Weiss
	Cameron and Trethowan
	Coon
	Fairbank
	Flotow
	Fragenheim
	Hackenbrock
	Nove-Josserand
	Ollier (2 cases)
	Wittek

ETIOLOGY

The etiology of Ollier's dyschondroplasia is, of course, unknown. The curious distribution of the lesions at the ends of the long bones suggested to Bentzon (1924) that the 'stripes' of cartilage followed the course of the branches of the nutrient artery. He injected the nutrient arteries of normal bones and found that such was indeed the case. From this he argued that probably the sympathetic nervous system was at fault. He then conducted a series of experiments on rabbits in which he destroyed part of the sympathetic chain by the injection of alcohol. Out of 27 rabbits successfully treated, 1 showed, in radiograms taken a month later, changes in one of the phalanges which resembled those in the phalanges in dyschondroplasia. However, histological examination in another four months revealed in these areas no cartilage. There was a considerable enlargement of the marrow cavity, and the cortex was of a coarser structure than that of the normal bone.

On the basis of these observations Bentzon argues that "Ollier's disease may be interpreted as the typical reaction of the bones against an active hyperæmia of the bone tissue, arising owing to anomalies in the vegetative nervous system." It seems to us, however, that the evidence is not sufficient to warrant the assumption of so unusual a departure from the habitual reaction of bone to hyperæmia. We do not mean that we feel the sympathetic nervous system can have no relation to the disease, but that it cannot cause it merely by producing hyperæmia. It is conceivable that it may exert in some other manner a direct influence on the metabolism of bone or cartilage.

In this connection there is another point of interest. In six of the cases of unilateral dyschondroplasia there has been a marked facial asymmetry (Bojesen, 1914, S. Johansson, 1916, C. Johannessen, 1923, Bentzon, 1924, J. W. F. Jansen, 1925, and Hessenthaler, 1929).

CASE REPORT

HISTORY—R J, male, aged 7 years (L H Reg No 31622/1932) He was born in London, the first child of Jewish parents, the only other pregnancy in the mother having been terminated by Cæsarean section because of heart disease There was no consanguinity either in the parents or in the grandparents At the age of 18 months he began to limp on the right leg, and was taken to a hospital for radiograms of bones The father was told that there was 1½ in shortening of the right femur, and that radiograms showed disease of the head and upper part of the shaft of the right femur Osteitis fibrosa cystica was suspected Radiograms revealed changes in other bones on the right side of the body On March 26, 1931, a portion of the lower end of the right tibia was excised for histological section No family history of bone disease was obtained (fifteen members of three generations on both sides of the family were questioned)

ON EXAMINATION—Well-nourished boy, of normal mental development Weight 3 st 9 lb Height 3 ft 9 in Mucous membranes normal colour, sclerotics white Teeth a few carious molars, upper central incisors and lower central and lateral incisors show serrated margins, no wedge-shaped teeth Pupils equal, circular, react to light and on accommodation Ocular movements full Fundi normal No abnormality in the cranial nerves, or elsewhere in nervous system Pulse regular, 70 Heart and lungs normal Blood-pressure 110/75 mm of mercury Abdomen normal No facial asymmetry Index fingers, tarsus, and carpus on both sides normal Skull, spine, thorax, and both upper limbs normal Right femur is shorter than left, with an outward bend (Fig 356) Measurements anterior superior iliac spine to internal malleolus, 22 in right, 24 in left Anterior superior iliac spine to top of patella, 11 in right, 12½ in left Bryant's triangle measurements, 1½ in right, 2 in left, that is, there is ½ in deficiency in neck of femur, indicating coxa vara Lower border of patella to internal malleolus, 9 in right, 9½ in left Sternoclavicular joint to bend of wrist, 18½ in right, 18½ in left Limitation of abduction with increased adduction in right hip Rotation tests normal Slight increased mobility in right ankle-joint Both knee-joints are loose Radiograms skull, spine, ribs, and all bones on left side of body normal Lesions seen in the following bones phalanges of hands, ilium, ischium, pubis, femur, tibia, fibula, metatarsals, and phalanges of foot (Figs 358-362) The most severe changes seen in the lower half of the femur, and upper third of the tibia dense longitudinal bony trabeculae with small, pale, mottled areas and scattered dense, punctate spots The whole pattern of the spongiosa is altered, and the corticalis is in some places thin and expanded Upper end of femur, lower end of tibia, upper ends of humerus, metatarsals, metacarpals, and phalanges show similar but less marked changes The largest pale areas measure 1.5 by 0.8 cm, and the smallest 0.1 cm across The crest of the ilium shows pale fan-like striations extending from the periphery Controlled radiograms showed that outside these areas the shadows of all bones examined were of normal density Blood chemistry serum calcium 10.5 mgrm per 100 c.c., plasma phosphorus 3.6 mgrm per 100 c.c., plasma phosphatase 0.335 mgrm per 100 c.c., red cells 5,700,000 per c.mm., hæmoglobin 96 per cent, colour index 0.84, Blood-count

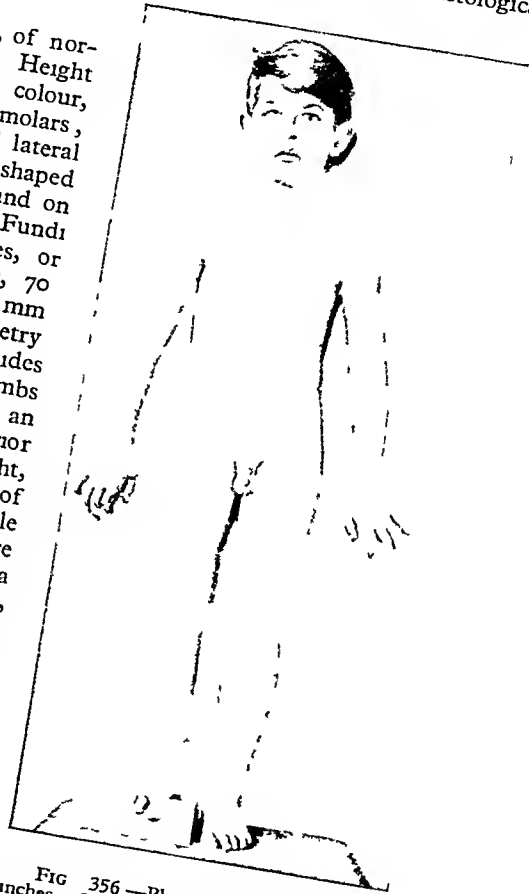


Fig 356—Photograph showing two inches of shortening in right lower limb Upper limbs, thorax, spine, and skull normal No facial asymmetry

white cells 10,000 per c mm, reticulocytes 0.6 per cent. Differential count normal. Urine normal, Bence-Jones protein not detected. Stools pale brown, total fat 4.2 per cent of dried faeces. On a known low calcium intake (0.39 gm), the calcium output in the urine (0.13 gm) and the faeces (0.24 gm) was normal.

HISTOLOGICAL REPORT (Professor H. M. Turnbull)—Tissue removed from lower end of right tibia, received from the Bland-Sutton Institute of Pathology, the Middlesex Hospital.

I have examined the hæmatoxylin-eosin and van Gieson sections. The bone has been broken in preparation, and it is difficult to be certain of the orientation. The corticalis contains a large piece of hyaline cartilage of irregular shape, upon which bone has been deposited in many places (Fig. 357). The cells are larger and more irregularly distributed than in normal epiphyseal cartilage. The cartilage is calcified in parts of its periphery. In such spots many of the cells are hypertrophic, but I cannot find the typical changes that are provisional for endochondral ossification. In parts of its periphery the cartilage passes directly into a zone of dense fibrous tissue. There appear to be other pieces of hyaline cartilage within the fibrous periosteum.

Cartilage is formed in focal osteitis fibrosa, at any rate in young subjects. There is no trace, however, of osteitis fibrosa here. The inclusion within the corticalis of remnants of atypical epiphyseal cartilage is a characteristic of congenital chondrodysplasia. I have not, however, in that condition seen a fibrous zone upon parts of the surface of remnants,

nor have I seen portions of cartilage within the fibrous periosteum as in this section. Otherwise, the section suggests congenital chondrodysplasia to me. There is considerable porosis of the corticalis but no evidence of rickets or malacia.



FIG. 357.—Photomicrograph of tissue removed from lower end of right tibia, received from the Bland-Sutton Institute of Pathology, the Middlesex Hospital. The section shows a large piece of hyaline cartilage of irregular shape upon which bone has been deposited in many places (see in obj.).

RADIOLOGICAL APPEARANCES

In every difficult case of disease of the ends of the long bones or of the bones of the hand or foot, radiograms of the entire skeleton must be taken, otherwise a case of dyschondroplasia may be overlooked. The disease may be limited to one bone only, to one limb, to one side of the body, or almost every bone may be affected. It is probable that the spine and the carpal and tarsal bones escape entirely, whilst the ribs, sternum, and skull are involved only very rarely.

The long bones—namely, humerus, radius, ulna, femur, tibia, and fibula—are those in which the typical appearances of dyschondroplasia are to be seen. The more rapidly growing end of a bone is that most commonly affected, but the opposite end may be involved too. The centre of the shaft remains normal except in very severe cases. The appearance of an affected region varies as the disease progresses. The changes seen in the early stages are well shown in the lower end of the femur and the upper end of the tibia of our case (see Figs. 359, 360). There is a translucent pyramidal area with its apex at the nutrient foramen and

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its base resting on the epiphyseal line This area is divided up by septa about 0.5 to 1.0 cm apart, running approximately in the long axis of the bone The lateral view may show the depth of involvement to be much less than might be expected from the antero-posterior pictures (C. Johannessen, 1923, Bentzon, 1924) Such translucent areas may extend over the whole width of the bone or be limited to any part of it (Burchard, 1913) At times there is only one narrow stripe in the middle of the bone



FIG 358 —Radiogram of pelvis Fan-like striation in crest of right ilium Pale striation, mottled areas, and scattered dense punctate spots in head and neck of right femur Bones on left side normal

The affected ends of the diaphysis may have an outline of normal size and shape, or alternatively they may be broadened This broadening tends to be a fusiform enlargement (Cleveland, 1928, Ballin, 1933), but in rarer cases it resembles that in diaphysial aclasis (Cole, 1926) Similarly small exostoses may occur near the ends of the diaphysis, but they are irregular in shape (Kummer, 1918, Weber, 1920, Weiss, 1923) and have a core rather than a shell of cartilage At the upper end of the femur the region usually affected earliest is the lesser trochanter (see Fig 358), and progressive involvement takes place across

the bone to the great trochanter (Bojesen, 1914, C Johannessen, 1923, Lindstrom, 1924) Sacerdote described a case in 1931 where only the great trochanter was involved

The upper end of the humerus is affected above the level of the insertion of the deltoid, but the striping is not here well marked When the lower end of the radius is involved it usually shows some shortening and forward bowing, at times resembling that to be seen in diaphysial aclasis or in Madelung's deformity The lower end of the humerus and the bones of the forearm of our case, however, are practically normal



FIG 359—Radiogram of femora. Right femur two inches shorter than left. Pale and dense streaks, pale mottled areas, and dark punctate spots in lower end of right femur. Extensive alteration in pattern of corticis and spongiosa. Left femur normal.

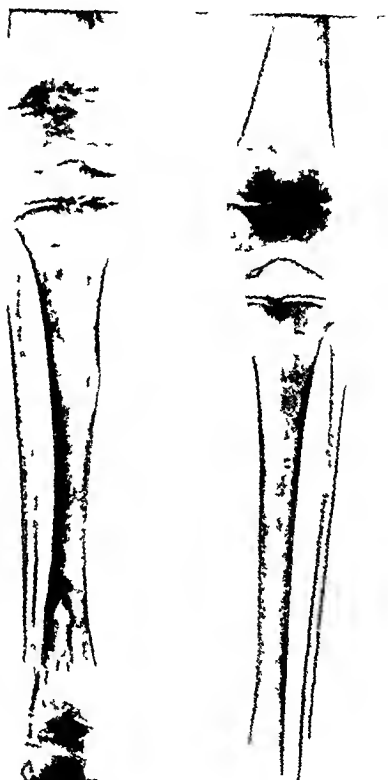


FIG 360—Radiogram of tibiae. Extensive areas showing pale striped and mottled areas at both ends of right tibia and near centre of shaft. Many dark punctate spots. Lines of cessation of growth are seen near both knee-joints, but the left tibia is otherwise normal.

In the ilium the distribution of the cartilaginous areas is most striking. Only the periphery of the ilium, usually over a fan-shaped area in the middle of the bone, is affected, and the striping is very marked (see Fig 358). In a few cases the whole of the flat portion of the bone is involved (Brockman, 1931, Kochs, 1932).

The ischium and the pubes, especially near the ischial tuberosity, may be affected, giving rise in the radiograms to a fluffy appearance due to the presence of many small rounded islands of cartilage. There is never any striping here.

The phalanges, the metatarsals, and the metacarpals differ from the long bones in that, as a rule, they contain only simple enchondromata which can in no way be distinguished from the isolated enchondromata occurring apart from dyschondroplasia. They may occur in any part of the diaphysis of the bone and grow to a considerable size. They are usually described as being of two types, central or medullary, and subperiosteal. In the absence of stereoscopic radiograms it is difficult to differentiate these types. Isolated enchondromata may also occur in any part of the shaft of the long bones. They are not common and cannot be regarded as typical of the condition. Occasionally, however, striping similar to that found in the long bones is seen in the metacarpals,

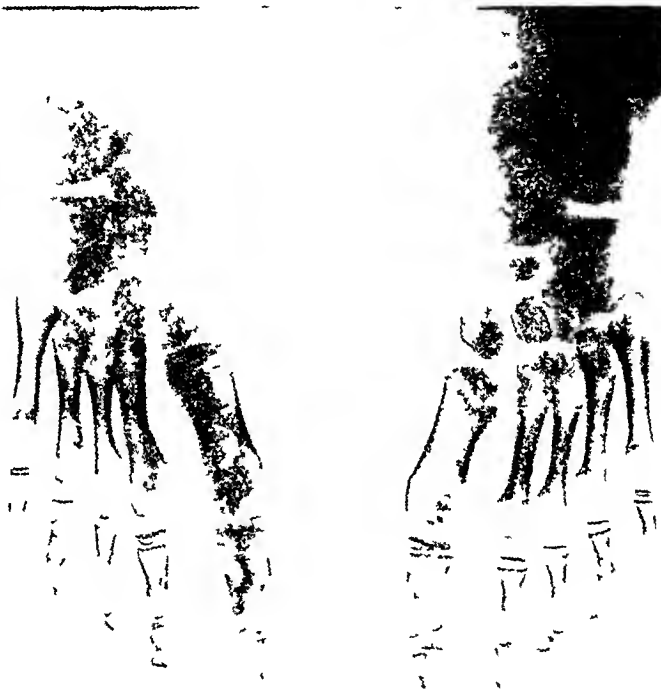


FIG 361—Radiogram of feet. Metatarsals and phalanges of right foot show small, pale, mottled areas and a few dense punctate spots. Left foot normal.

metatarsals, and phalanges. This occurred in the case of Huber and Advenier (1929), and is seen also in our case. Fig 362 shows the disease affecting the growing ends of some of the phalanges and metacarpals, in the second phalanx of the ring finger striping can be distinctly seen—in strong contrast is the sharply circumscribed, clear area in the middle of the second phalanx of the index finger, which resembles the enchondromata usually to be found in the fingers.

As the child grows older, the typical striped appearance disappears and is replaced by a speckling due to islands of dense calcification. As our own case is too young to show this well, we reproduce by courtesy of Mr A. Rocyn-Jones the right femur of a girl now aged 21 years who is suffering from dyschondroplasia and in whom nearly every bone is affected. A radiogram (Fig 363) taken

at the age of 3 shows the typical appearance described above for early cases. At the age of 15 marked speckling (*Fig 364*) has replaced the earlier appearances. The case is only introduced here to show the appearance of dyschondroplasia at a later age. The same appearance is well shown in published cases (Boinet and Stephan, 1903, Cole, 1926, Cleveland, 1928, Hcssenthaler, 1929). At a later

stage still there is a tendency for the structure of the bone to approach more closely to the normal, although it is doubtful if it ever becomes completely so. That speckling does not always occur, however, can be seen in the case of a woman aged 38 published by Weiss (1923). Here the striping has spread into the epiphysis, which can no longer be distinguished from the diaphysis.

The epiphyses are completely unaffected at birth. The exact age of their involvement varies in different cases. They seldom show the striping of the long bones, but present the speckled appearance due to irregularities of calcification. This is just beginning to show in the femoral epiphysis of our case (*see Fig 359*). If only a small strip of the diaphysis is involved, then only an approximately corresponding width of epiphysis seems to suffer (J W F Jansen, 1925). After the age when the epiphysis and diaphysis normally unite the appearance of the two becomes uniform and shows irregular speckling (Cole, 1926).



FIG 362.—Radiogram of right hand. Right radius and many phalanges and metacarpals show pale mottled areas, sometimes with dense punctate spots.

DIFFERENTIAL DIAGNOSIS

Dyschondroplasia and the conditions which resemble it are rare. Since neither the physical nor the biochemical findings serve to identify the disease, the diagnosis largely depends upon examination of radiograms. In an early clear-cut case the picture is so typical that it should present no difficulty. Nevertheless, confusion not infrequently occurs, even in obvious cases. Thus Ballin (1933) writing on 'parathyroidism' describes the radiograms in what is clearly a well-marked case of dyschondroplasia as showing "the simultaneous existence of osteoporosis and osteitis fibrosa cystica."

In cases which are unilateral or nearly so, no difficulty is likely to arise. Unilateral diaphyseal aclasis does occur. Stocks and Barrington (1925) found

evidence of this in 11 per cent of 446 cases, but in this series radiograms of the whole skeleton were not available. We know of only one case of multifocal osteitis fibrosa where the distribution was unilateral (Fairbank, 1933)

The positive diagnosis of dyschondroplasia is made on three main points —

- 1 The onset is in early childhood
- 2 Radiograms show changes limited to the ends of the long bones. There is striping of rarefied areas. The epiphyses are involved only secondarily. Speckling appears in the affected metaphyses and epiphyses as growth proceeds
- 3 A portion of tissue corresponding to a pale area in a radiogram is found, when excised and examined histologically, to consist of cartilage

Diaphysial Aclasis (Hereditary Multiple Exostoses).—This is characterized by four main features which differentiate it from dyschondroplasia



FIG 363—Radiogram of lower limb in a child (aged 3) suffering from dyschondroplasia. Patient is now 21 years of age and nearly every bone is affected. Both lower end of femur and upper end of tibia show characteristic translucent areas divided up by septa running approximately in the long axis of the bone.

FIG 364—Radiogram of lower end of femur taken twelve years later than that in Fig 363, the patient being 15 years of age. Speckling due to islands of dense calcification has replaced the striping found at the earlier age.

1 Heredity is a very striking feature. Stocks and Barrington (1925) found that in 1124 recorded cases 727 or 64.7 per cent gave a definite history of the disorder occurring among antecedents or relatives.

2 The presence of ossifying enchondromata (exostoses) is almost invariable. They are usually pedunculated, may arise anywhere along the length of the bone, point away from the end of the bone, and are well defined and clear-cut. In dyschondroplasia they arise near the epiphyses, point in any direction, are rough and irregular, and never reach any great size (Ehrenfried, 1917).

3 Broadening of the metaphysis is essential to the diagnosis of diaphysal aclasis. It is a fairly regular process, beginning abruptly with a sudden trumpeting of the shaft and with sides approximately parallel. In dyschondroplasia the metaphysis is often normal in size and shape but it may be expanded in a fusiform manner. Only very occasionally does it approach in appearance the uniform trumpeting described above (Keith, 1920).

4 In diaphysal aclasis, although there may be enchondromata present, there is fairly regular ossification giving a homogeneous structure to the bone. In dyschondroplasia in the early stages there is regular arrangement of cartilage and in the later stages stippling of the epiphyses.

There are, of course, a few cases which it is difficult to place in any group (Voorhoeve, 1924, Mauclore, 1926, Dahl, 1930), but this is probably because they have only been observed in one stage of their progress. If they could be watched growing up from infancy there would be less difficulty.

Multiple Enchondromatosis—This is a condition affecting the long bones of the hands and feet. The radiographic appearances are identical with those seen in the hands and feet in dyschondroplasia. It is impossible to say whether or not the former disease is a localized form of the latter. It is equally impossible to explain why it is that the hands and feet in cases of dyschondroplasia so often show changes which are different from those in the rest of the skeleton. There are rare cases (Steudel, 1892, Fragenheim, 1911, Hackenbrock, 1922) in which massive enchondromata occur in the hands and feet, giving rise to hideous deformity. This probably occurs only in association with dyschondroplasia. It is difficult to classify accurately those cases which are published under the title "systematized multiple enchondromata", but they seem for the most part to be atypical cases of dyschondroplasia.

Voorhoeve's Disease—The radiograms of bones show long fine parallel lines of increased density in the region of the metaphyses and some mottling of the epiphyses. The distribution is similar to that in dyschondroplasia, but the pale shadows of cartilaginous areas are missing (Voorhoeve, 1924).

Speckled Epiphyses—The small bones of the hand and foot and the epiphyses of the long bones appear in radiograms as countless small black dots (Fairbank, 1927).

Osteopoikilosis.—X-rays show areas of excessive density scattered throughout the entire bone. These areas are usually a few millimetres in diameter, and may be rounded or elongated to a few centimetres in length, in which case the long axis is in the long axis of the bone (Albers-Schonberg, 1915, Ledoux-Lebard, 1917).

Generalized Osteitis Fibrosa (Hyperparathyroidism).—The diagnosis of this condition by no means depends only on radiograms. The patient may show generalized tenderness of bones, and there may be symptoms unrelated to the skeleton, such as vomiting, renal colic, or muscular hypotonia. The characteristic biochemical findings of hyperparathyroidism will be present—namely, a persistently high serum calcium and low plasma phosphorus. The output of calcium in the urine may reach six times the normal figure. Radiographic examination of the bones by the controlled method will show diminished density of the shadows of all bones affected. In addition to this generalized change, multiple pale cyst-like areas may be found anywhere in the skeleton, and in the case of the long bones there may be expansion of the corticulis (Mandl, 1929, Barr, Bulger, and Dixon, 1929, Wilder, 1929, Hunter and Turnbull, 1931, Gordon-Taylor and Wiles, 1932).

Focal Osteitis Fibrosa.—In cases of focal osteitis fibrosa with multiple foci a large number of bones in the body may be affected. In spite of this, the serum calcium, plasma phosphorus, and urinary calcium excretion remain normal (Hunter, 1930). There is no generalized rarefaction of the skeleton, many areas of bone being found which compare in density to the control. The distribution of the cystic areas is quite haphazard. The areas of bony rarefaction show irregular shapes. They are often limited by a line of increased density and they may expand the corticalis. In dyschondroplasia the pale shadows of cartilaginous areas are localized to the neighbourhood of the metaphyses.

Gaucher's Disease—In 1922 Pick discovered a gross osseous form of this disease. He described cases with marked skeletal changes in the form of erosion of the corticalis, pathological fractures, and angular curvature of the spine. Junghagen in 1926 called attention to the use of radiograms of bones in the diagnosis of Gaucher's disease. In view of the obvious enlargement of the spleen and the changes in the blood, confusion with dyschondroplasia should not occur.

Xanthomatosis of Bones.—Xanthomatous deposits in the skull, especially in association with exophthalmos and diabetes insipidus, are now well known (Hand, 1893, Schuller, 1915, Christian, 1919). That the calvaria may escape, though other bones are extensively affected, has been shown recently by Snapper and Parisel (1933). In some cases increase in the total cholesterol content of the blood confirms the diagnosis. Histological examination of a portion of excised bone shows the characteristic foam cells of lipid-granulomatosis.

Lymphangioma of Bone.—There is one remarkable case (Wrede, 1911) which might well have been mistaken for dyschondroplasia on radiographic evidence alone. Pale shadows were observed in the upper ends of the tibia and fibula of a girl aged 6 years. Biopsy was refused, but the appearance and history of the child suggest that the bone lesions may have been lymphangiomata.

COURSE

The actual age of onset usually is not known, but some abnormality is often first noticed between the first and second years of life, when, as a rule, one limb is found to be shorter than its fellow. The difference in length becomes progressively greater as growth proceeds. By about six years of age bending of the limbs or enlargement of one or more fingers may be apparent. Deformity may be due to weight-bearing causing bending of the bone, to the irregular rate of growth in those cases in which only a portion of the width of the diaphysis is affected, or to the different rate of growth where only one of the paired bones is affected.

At the earliest stage the diaphysis only is involved and the epiphyses are unaffected. After about five years of age the epiphyses may become involved. The radiographic appearances suggest that the disease spreads from the diaphysis to the epiphysis and it may well be that this spread bears some relation to a change in blood-supply. However, we have at present very little exact knowledge as to the details of the blood-supply to the epiphyses at different ages.

As the patient gets older, areas of calcification appear in the affected areas, giving to radiograms a very typical appearance. This change probably occurs round small patches of degeneration in the cartilage. The age at which it takes place is subject to great variation but it does not usually commence until after six

years and it may be delayed much longer. By the time the epiphyses would normally have united to the diaphysis the two present a uniform picture and appear to consist of cartilage studded more or less thickly with calcified areas a few millimetres across. It is probable that the bones never become completely normal, but some of the radiograms of the older cases show an approach to the normal structure, the 'speckling' becomes much fainter and some cancellous structure appears.

PROGNOSIS

The majority of cases reported in the literature are children, and no mention of the same cases appears subsequently. There are, however, several cases reported in their early twenties as alive and well, and the patient described by Weiss (1923) reached the age of 38 before attending a clinic, but he does not say of what she then complained. The case reported by Boinet and Stephan (1903) died at the age of 35, apparently from a sarcoma, but there had been several fractures and also some operative interference. The patient described by Nehr Korn (1898) was probably a case of dyschondroplasia and he died of a sarcoma of the thigh at the age of 49 years. In the case reported by Lea (1929) a frontal glioma appeared at the age of 28 years. The patient described by Speiser (1925) died of anæmia at the age of 4½ years. The patient reported by Nasse (1895) developed massive tumours of the fingers and thigh, and eventually was reported by Fragenheim (1911) as having died following a disarticulation of the hip performed for the relief of pain.

The actual incidence of sarcoma cannot be determined, but the prognosis apart from this seems by no means bad. Most patients seem to reach adult life, when their symptoms are mainly those of their deformities and sometimes of a secondary arthritis.

TREATMENT

Treatment is concerned with the prevention and relief of deformities and proceeds along the usual orthopædic lines. Osteotomy has been performed in at least six cases (Kohler, 1905, Thiemann, 1910, Hackenbrock, 1922, J. W. F. Jansen, 1925, Cleveland, 1928, Flotow, 1929) and has met with no untoward results. Fractures are of fairly common occurrence and, like the osteotomies (Kohler, 1905), appear to unite well.

It is with much pleasure that we thank Professor H. M. Turnbull, Professor J. McIntosh, Mr G. Gordon-Taylor, Mr A. Rocyn-Jones, Dr N. S. Finzi, Dr M. H. Jupe, Dr L. J. Rac, and Dr D. Mintzman, for their help in the investigation of this case.

BIBLIOGRAPHY

- ADAMS, J. E., *Proc. Roy. Soc. Med. (Sect. Dis. Child)*, 1918-19, **xii**, 5.
 ALBERS-SCHONBERG, *Forts. a. d. Geb. d. Röntgenstrahlen*, 1915-16, **xviii**, 174.
 ASHHURST, A. P. C., *Ann. of Surg.*, 1916, **lxi**, 167.
 ANSCHUTZ, *Munch. med. Woch.*, 1908, **lv**, 1717.
 BALLIN, M., *Jour. Bone and Joint Surg.*, 1933, **xv**, 120.
 BARR, D. P., BULGER, H. A., and DIXON, H. H., *Jour. Amer. Med. Assoc.*, 1929, **xcii**, 951.
 BENTZON, P. G. K., *Acta Radiol.*, 1924, **iii**, 89.

- BOINET and STEPHAN, *Arch gen de Med*, 1903, cxc1, 449
- BOJESSEN, A, *Hosp-tid Kobenh*, 1914, ii, 1017, 1059
- BROCKMAN, E P, *Proc Roy Soc Med (Sect Orthop)*, 1931, xiv, 34
- BROMER, R S, and RUTHERFORD, J L, *Amer Jour Roentgenol*, 1931, xvi, 428
- BURCHARD, A, *Forts a d Geb d "*, 1913, xii, 113, 291
- CAMERON, H C, and TRETHOWAN, *Soc Med (Sect Dis Child)*, 1917-18, xi, 45
- CHRISTIAN, H A, *Defects in Membranous Bones, Exophthalmos and Diabetes Insipidus, Contributions to Medical and Biological Research*, 1919, i, 390 New York Paul B Hoeber, Inc
- CLEVELAND, M, *Surg Gynecol and Obst*, 1928, xlvii, 338
- COLE, W H, *Ibid*, 1926, xli, 359
- COON, C E, *Amer Jour Orthop Chir*, 1911-12, ix, 604
- DAHL, B, *Acta Orthop Chir*, 1930, i, 127
- EHRENFRIED, A, *Jour Amer Med Assoc*, 1917, lxxviii, 502
- FAIRBANK, H A T, *Brit Jour Surg*, 1927-8, xv, 127
- FAIRBANK, H A T, Personal communication, 1933
- FLOTOW, F, *Zeits f orthop Chir*, 1929, li, 505
- FRAGENHEIM, P, *Beitr z klin Chir*, 1911, lxxiii, 226
- GORDON-TAYLOR, G, and WILES, P, *Brit Jour Surg*, 1931-2, xiv, 606
- HACKENBROCK, M, *Arch f orthop u Unfall Chir*, 1922-3, xxi, 206
- HAND, A, *Arch of Pediatrics*, 1893, v, 673, *Amer Jour Med Sci*, 1921, clvii, 509, *Proc Philadelphia Pathol Soc*, 1891-2, xvi, 282
- HESSENTHALER, M, *Forts a d Geb d Rontgenstrahlen*, 1929, xxxix, 645
- HUBER, J, and ADVENIER, W, *Arch med des Enfants*, 1929, xxxii, 595
- HUNTER, D, *Lancet*, 1930, i, 953
- HUNTER, D, and TURNBULL, H M, *Brit Jour Surg* 1931-2, xiv, 203
- JANSEN, J W F, *Acta Radiol*, 1925, iv, 133
- JANSEN, M, *Robert Jones Birthday Volume*, 1928, p 43 London Oxford Medical Publications
- JOHANNESSEN, C, *Norsk Mag f Læge*, 1923, lxxiv, 629
- JOHANSSON, S, *Zentralb f Chir*, 1916, xlii, 864
- JUNGHAGEN, S, *Acta Radiol*, 1926, v, 506
- KEITH, A, *Jour of Anat* 1920, liv, 101
- KOCHS, J, *Arch f orthop u Unfall Chir*, 1932, xxxii, 419
- KOHLER, A, *Die normale u pathologischen Anatomie d Hufsigelenks u Oberschenkels*, 1905, p 132 Hamburg Grafe and Sillem
- KUMMER, E, *Rev med de la Suisse Rom*, 1918, xxxviii, 569
- LEA, A, *Frankf Zeits f Pathol*, 1929, xxxvii, 336
- LEDOUX-LEBARD, R, CHABANEIN, and DESSANE, *Jour de Radiol et d'Electrol*, 1917-18, ii, 133
- LINDSTROM, L J, *Acta chir Scand*, 1924, lviii, 190
- MANDL, F, *Zentralb f Chir*, 1926, lvi, 260, 1929, lvi, 1739
- MAUCLAIRE, *Rev d'Orthop*, 1926, xiii, 653
- NASSE, D, *Sammlung klin Vortr*, 1895, Chir No 34, 209
- NEHRKORN, A, *Beitr z klin Chir*, 1898, xxii, 800
- NOVÉ-JOSSERAND, G, *Bull Soc Chir Lyons*, 1899, iii, 40
- OLLIER, *Ibid*, 22
- PICK, L, *Med Klin*, 1922, xviii, 1408
- RICHARD, H, and DUPUIS, P, *Rev d'Orthop* 1933, x, 662
- SACERDOTE, G, and BONOMINI, B, *Chir degli Org di Movimento*, 1931, xvi, 207
- SCHULLER, A *Wien med Woch*, 1921, lxxi, 510, *Forts a d Geb d Rontgenstrahlen*, 1915-6, xxi, 12, *Brit Jour Radiol*, 1926, xxi, 156
- SNAPPER, I, and PARISEL, CH, *Quart Jour Med*, 1933, NS ii, 407
- SPEISER, F, *Virchow's Arch*, 1925, cclviii, 126
- STEUDEL, *Beitr z klin Chir*, 1892, viii, 503
- STOCKS, P, and BARRINGTON, A, *Treasury of Human Inheritance*, 1925, iii, 54
- THIEMANN, H, *Forts a d Geb d Rontgenstrahlen*, 1910, xiv, 84
- THOMASON, T H Cited by Bromer, R S, and Rutherford, J L
- VALENTIN, B, *Vcrh d zite Kongress d deut orthop Ges*, 385
- VOORHOEVE, N, *Acta Radiol*, 1924, iii, 407
- WEBER, E P, *Brit Jour Dis Child*, 1920, xvii, 85
- WEISS, K, *Forts a d Geb d Rontgenstrahlen*, 1923, xxxi, 615
- WILDER, R M, *Endocrinology*, 1929, viii, 231
- WITTEK, A Cited by Fragenheim, P
- WREDE, L, *Beitr z klin Chir*, 1911, lxxiii, 213

COLOSTOMY

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THE chief value of colostomy as a palliative operation in malignant disease of the rectum is afforded by the relief of obstruction, and to a varying degree by the relief of important and troublesome symptoms such as diarrhoea, tenesmus, pain, bleeding, and discharge. The improvement in these symptoms varies greatly with different patients, as much owing to differences in temperament and management as to differences in the types of primary tumour (scirrhus, colloid, ulcerating, or vascular), their situation (upper, middle, or lower third of the rectum), and the extent of extra-rectal spread.

In *some* cases, such as those with obstruction, recto-vaginal, recto-vesical, or vesico-colic fistula, the relief given by a colostomy is undeniable, in *most* cases after colostomy there is at first, for a varying number of months until a late stage of the disease is reached, a definite improvement not only locally, but also in the patient's general condition, this is to be accounted for by the ability to take a more normal diet, by proper bowel evacuations, relief of rectal irritability, and more regular sleep. For these reasons colostomy is rightly regarded as a beneficent operation for the relief of inoperable rectal cancer.

The benefits conferred by a colostomy are most appreciable if the operation is done at a stage when the patients are still capable of some general improvement, and have time and courage to adapt themselves to a colostomy life. If the operation is not done until a late stage of the disease, the operative risks are greatly increased: the patients are often in a poor state of health, under-nourished, dehydrated, and worn out by loss of sleep from diarrhoea, tenesmus, or pain. Accompanying conditions such as dental sepsis, chronic bronchitis, and urinary infection, retention, or failure, are often present and aggravate the risk of an abdominal operation.

The following study concerns a series of 500 cases of palliative colostomy for inoperable rectal cancer which have been admitted into St Mark's Hospital in the course of the last twenty-four years. We shall show that the operation mortality of colostomy in these advanced cases is considerably greater than that of colostomy in a parallel series of 470 operable cases in which an excision of the rectum was either planned or carried out as a second-stage operation. The added risks of palliative colostomy when done in advanced age or in the late stages of obstruction will be shown, but even so we believe that the operation is justified by the relief of obstruction and other symptoms, and in certain cases by a notable prolongation of life.

It is difficult, however, to assess in a satisfactory manner the value of colostomy in a series of cases. The relief of symptoms and the prevention of obstruction

cannot be measured in words or tables. The definite facts which can be recorded are concerned chiefly with (1) Operation mortality in relation to age, sex, and stage of the disease, (2) The causes of operative deaths, with suggestions for avoiding certain immediate complications, (3) The duration of life after colostomy, (4) Late complications and sequelæ connected with colostomy, (5) Operative technique of left iliac colostomy.

OPERATION MORTALITY AND IMMEDIATE POST-OPERATIVE COMPLICATIONS

The following statistics and discussion of operative mortality after colostomy and immediate post-operative complications are compiled principally from 500 cases of palliative colostomy for inoperable cancer of the rectum and pelvic colon, in which series 67 deaths occurred (13.4 per cent).

Table I—OPERATIVE MORTALITY OF COLOSTOMY IN RELATION TO AGE AND SEX

AGE INCIDENCE IN YEARS	MALES			FEMALES		
	No Operated Upon	No of Operative Deaths	Per Cent	No Operated Upon	No of Operative Deaths	Pe Cent
20-29	7	1	—	4	0	—
30-39	12	0	—	11	0	—
40-49	37	7	19	13	1	7.7
50-59	113	13	11.5	39	5	12.5
60-69	151	15	10	47	12	25
70-79	53	10	19	12	3	25
80-89	1	0	—	—	—	—
Total	374	46	12	126	21	17

In 236 cases under 60 years of age there were 27 operative deaths = 11.4 per cent

In 198 cases between 60 and 69 years of age there were 27 operative deaths = 13.6 per cent

In 66 cases of 70 years and over there were 13 operative deaths = 20 per cent

A consideration of *Table I* shows the following points —

- 1 The preponderance of males over females in the proportion of 3 to 1
- 2 The higher proportion of inoperable carcinoma in females under 40 years of age (12 per cent) compared with males under 40 years of age (5 per cent)
- 3 In males 14 per cent were over 70 years of age, but in females only 9 per cent
- 4 The total mortality as shown is 13.4 per cent, with the higher mortality of 17 per cent in females compared with 12 per cent in males. The female deaths are seen to rise steadily in each decade from 40 onwards, but in males the mortality has been irregular. It was at its maximum in the decade 40-49 and in patients over 70, in the former group there occurred 4 deaths from peritonitis which will be further considered in the following section, and in the latter group the principal causes of death have been pulmonary or cardiac complications.

Among a separate series of 470 cases which were assessed as "operable" there occurred 12 deaths (2.5 per cent) which were directly attributable to the colostomy.

These may now be added to the 67 deaths recorded in *Table I*, giving thus a total of 79 operative deaths from colostomy, an analysis of which is shown in *Table II*. We wish to draw particular attention to the deaths from peritonitis and paralytic obstruction.

Table II—CAUSES OF 79 OPERATIVE DEATHS IN 970 CASES OF COLOSTOMY, 1910-33

CAUSE OF DEATH	TOTAL NUMBER OF DEATHS DUE TO THIS CAUSE	MALES	FEMALES
Heart failure	11	7	4
Pulmonary complications	11	8	3
Pulmonary embolism	2	1	1
Peritonitis	12	7	5
Paralytic ileus	9	6	3
Mechanical obstruction	5	4	1
Toxæmia from pre-operative obstruction	4	3	1
Prolapse of small intestine	9	7	2
Uræmia	6	4	2
Cachexia	5	2	3
Miscellaneous	5	4	1
Total	79	53	26

Cardiovascular and Pulmonary Complications—This forms the largest group, with a total of 24 deaths. There were 11 deaths from cardiac failure, the average age of these cases being 66 years, at an average time of five days after operation. In one case the rectal carcinoma had been assessed as operable, but the patient collapsed suddenly on the ninth day and died in a few minutes; a post-mortem examination revealed atheroma of the coronary arteries, with occlusion of one branch; the remaining patients had inoperable growths and died from myocardial or coronary disease. There were 10 deaths from bronchitis or bronchopneumonia at an average age of 64 years, and an average time of ten days after operation, and 1 death from acute pulmonary œdema within twenty-four hours of operation in a young man of 25 with an advanced obstructing carcinoma in the upper third of the rectum. There were 2 deaths from pulmonary embolism—a male, aged 64, on the fifth day, and a female, aged 62, on the fourth day—in both cases secondary deposits were proved to be present in the liver.

Peritonitis.—The 12 cases that died from peritonitis after colostomy form an instructive group, in 10 of these cases the presence of peritonitis was proved either at a secondary operation or post mortem, and the causes of the infection may be classified as follows—

1 In 3 cases the peritonitis was secondary to suppuration in the abdominal wall round the colostomy, and death took place on the thirteenth, thirteenth, and fourteenth day after operation respectively. In each case the 'delayed' method of opening the colostomy forty-eight hours after operation had been adopted.

2 In 5 cases abdominal exploration of the rectal growth must be accounted the exciting cause of peritonitis—the following are the significant details of these cases—

Case 1 —E W, a gardener, aged 49, with an inoperable carcinoma at the recto-sigmoid junction which had been explored at another hospital a few weeks previously. Laparotomy revealed a large fixed mass in the lower pelvic colon with dense surrounding adhesions, and a left iliac colostomy was established. Peritonitis with obstruction supervened, and jejunostomy with drainage of the pelvis was done on the fourth day, but the patient died next day.

Case 2 —T M, a porter, aged 45, with a small mobile carcinoma at 15 cm from the anus. The tumour was explored through a paramedian incision and left iliac colostomy was performed. On the second day the patient had a rapid pulse and moderate abdominal distension, this became worse, and a caecostomy was done, but the patient died from peritonitis on the fourth day. At the post-mortem no definite focus of infection was found.

Case 3 —Mrs A E, aged 58, with a large fungating carcinoma in the upper third of the rectum. An exploratory laparotomy and a left iliac colostomy was done, on the second day the abdomen became distended, small amounts of flatus were passed per colostomy, but a condition of partial obstruction developed. On the sixth day, under spinal anaesthesia, a further laparotomy was done and revealed a low-grade peritonitis, many coils of small gut being adherent, and flakes of lymph present. Death took place the same evening, and at the post-mortem a general peritonitis was confirmed, without any definite focus of infection being found. Secondary deposits were present in the diaphragm and right lung.

Case 4 —E C, a carman, aged 52, with an extensive carcinoma in the middle third of the rectum. There was a previous history of dysentery. A paramedian laparotomy was done, and some adhesions between the growth and the bladder were broken down with a finger before establishing a colostomy. On the third day vomiting and hiccups began, and the patient rapidly failed and died on the same day. A post-mortem examination showed pelvic peritonitis with the small intestine adherent in the pelvis where adhesions between the bladder and the rectum had been separated, and a small perforation into the rectum was demonstrated at this point.

Case 5 —J R, a man, aged 63, with a constricting carcinoma at 12 cm from the anus. Laparotomy confirmed the presence of a suprapерitoneal growth, and an adherent loop of ileum was separated from the growth. A fistula into the ileum was then found to be present, and the affected loop was resected. A colostomy was then made and a Paul's tube tied in, but the patient died from peritonitis on the fourth post-operative day.

It will be noted that in the first three cases in this group no definite focus of the peritoneal infection was found. Presumably it arose from handling the growth or mobilizing adhesions in the neighbourhood of the growth, and this experience indicates the extreme care and gentleness which should be exercised in exploring a carcinoma of the pelvic colon or upper rectum through an abdominal incision. If the growth is inoperable, there is no object in breaking down adhesions except to the minimum extent necessary for mobilizing the pelvic colon for a colostomy. The presence of diverticulitis of the pelvic colon in association with a carcinoma is a dangerous one which calls for special notice of the risk of breaking down adhesions and exposing septic areas.

3 In 2 cases peritonitis was caused by the opened colostomy slipping back into the abdominal cavity, in one case owing to the rod being inadvertently removed on the second day, and in the other case the rod ulcerated through the spur on the eighth day after operation, allowing the lower end to slip back into the pelvis.

4 In one case a fatal peritonitis resulted from a perforation of the colon a few inches proximal to the colostomy.

Case 6 —The patient, Miss M C, aged 53, had a large inoperable rectal carcinoma. A colostomy was performed and was opened on the following day. Colostomy wash-outs were then begun in order to relieve her distension. On the second evening after operation acute abdominal distension rapidly developed after an ox-bile enema had been given into

the colostomy, with severe griping pain and a pulse-rate of 156 per minute. She died on the third day after operation, and at the post-mortem a general peritonitis was found originating from a perforation of the descending colon 4 in above the colostomy at the point where the bowel turned forwards from the posterior abdominal wall to the colostomy, at this point there was a small linear slit in the bowel wall about half an inch long.

It is believed that the perforation was caused by an injudicious attempt to pass a flatus tube round this 'corner' of the bowel, and point is attached to this theory in that this accident is known definitely to have caused the death of another patient not included in this series four days after an abdomino-perineal excision of the rectum. In this case a post-mortem examination revealed an exactly similar perforation which had undoubtedly been caused by a tube. The lesson which may be drawn from these cases is that under certain conditions a rectal tube may be dangerous for passing down a colostomy. In fat subjects the bowel is often thin-walled and friable, and a soft rubber catheter, about No. 16 in size, is the only kind which should be used for this purpose.

5. In one case operated upon in 1910 the pelvic colon was divided above an inoperable growth. The upper end was brought out as a terminal colostomy, the lower end being closed and dropped back. Symptoms of peritonitis began on the fourth day. Post-mortem septic peritonitis due to leakage from the dropped-back colon was found to be present.

Paralytic Ileus—In this group there were 9 patients, 6 males and 3 females, and all had symptoms of continued obstruction following the opening of the colostomy. These cases occurred at intervals from 1911 to 1933, and 4 of the cases were assessed as operable. In the 5 cases that have occurred since 1928 either post-mortem examination or a secondary operation has been performed, and we are satisfied that the condition was one of paralytic ileus involving the small intestine. Four previous cases have an identical history and have accordingly been placed in this group. General anaesthesia was used in all the cases but two, in which spinal anaesthesia was given.

The composite clinical picture in these cases is as follows. The colostomies were opened at an average time of approximately thirty-six hours after the operation, and the first symptoms and signs were those of slight abdominal discomfort and distension appearing from the second to the fourth day. Colostomy wash-outs were immediately commenced with relatively poor results, and abdominal distension gradually increased, being at times slightly reduced by wash-outs and finally failing to respond to any form of treatment. The usual hypodermic injections such as pituitrin and eserine were given in conjunction with various wash-outs, turpentine, ox-bile, and human bile, and also intravenous salines. Acetyl-choline was not used in any of the cases in this series as the majority occurred before this treatment was described. The following is a characteristic case—

Case 7—S. S., aged 55, a storekeeper, was admitted with an operable carcinoma in the lower third of the rectum. The patient was a thin, underfed subject. Examination of the heart revealed no abnormality, the blood-pressure was 175/125, and the blood-urea 67 mgrm per 100 c.c. of blood. Five days after admission a left inguinal colostomy was performed under spinal anaesthesia, there was very little intraperitoneal manipulation. The colostomy was opened twenty-four hours after the operation, there was a slight escape of flatus and the abdomen appeared quiet and was not distended. The onset of the trouble was first noticed eighty hours after the operation, when the patient vomited an aperient which had been given, and complained of slight dull abdominal pain. On examination the abdomen

was found to be slightly distended in all quadrants, the pulse-rate was 106, there was some tendency to hiccup, and on auscultation the abdomen was silent. A flatus tube was passed into the upper colostomy opening and a slight amount of flatus obtained. Following this, 1 c.c. of pituitrin was given hypodermically, and twenty minutes later an ox-bile colostomy wash-out was given, a faecal-coloured result being obtained with a slight amount of flatus. There was no further vomiting, the tongue was moist, the pulse remained above 100, and the patient was fairly comfortable except for slight abdominal discomfort due to the distension which was still present, the abdomen was still silent. At this stage 0.5 c.c. of pitressin was given three-hourly. Five to six hours later there had been no passage of flatus per colostomy, the pulse-rate was rising gradually, and the abdomen was still distended—slightly more so in the upper half, a Ryle's stomach tube was passed and 12 oz. of dark-brown acid contents withdrawn. This tube was kept *in situ*, and further stomach contents were withdrawn two-hourly. At the same time a continuous intravenous normal saline with 5 per cent glucose was given at the rate of 10 oz. per hour for the next seven hours, and then, twenty-two hours after the onset of symptoms, another ox-bile enema was given with a slightly faecal and good flatus result. The pulse-rate had now risen to 130 per minute, the blood-pressure was 160/120, but the abdomen was less distended. A second operation was being considered at this time, but became inadvisable as shortly after this the patient became delirious and then comatose. The abdomen remained in a condition of silent distension and the pulse-rate slowly increased. The patient finally died approximately forty-eight hours after the onset of vomiting—that is, on the fifth day after the operation.

Laboratory tests showed the urinary chlorides to be greatly diminished and later absent, despite the administration of intravenous chlorides, the blood-urea rose steadily, suggesting a uræmic origin to the condition, however, the van Slyke clearance tests performed in this case clearly showed that despite the rising blood-urea the kidney function was unimpaired, and urine with a very high urea concentration continued to be excreted.

At the post-mortem a distended condition of the gastro-intestinal tract was found involving the stomach and the whole of the small intestine to within 2 ft. of the ileocaecal valve, at this point the distension gradually faded, and the remaining small gut distal to this point and the colon were found to be collapsed. There was no peritonitis or free fluid, and no adhesions were present in any part of the abdomen, there was no abnormality in any of the other organs.

In three other cases in which a post-mortem examination was performed, and in one other in which a second laparotomy was done, a similar condition with no evidence of peritonitis was found. Of the cases in this series, 3 were simple colostomy operations with relatively little intraperitoneal manipulation, and the remaining 6 had additional laparotomy wounds with more extensive abdominal manipulation. In 4 cases second operations were performed in a fruitless attempt to relieve the condition, though one case survived for nine days after a jejunostomy had been performed.

With regard to the causation, the only explanation which we have to offer is that this complication took place in patients with a poor bowel tone in which early distension took place after operation and *before the colostomy was opened*. We think it is correct to say that in the early cases in this series the abdominal wall was closed more loosely round the colon with the deliberate intention of allowing flatus to pass round the loop. In recent years the practice has been to make a rather tighter-fitting colostomy and to close the abdominal wall in layers as snugly as possible, so that until opened the bowel is actually obstructed.

This view is borne out by the figures, which show that 5 of these cases have occurred since 1928 and also that complications such as prolapse of omentum and small gut through the colostomy wound have in later years become much less frequent. We think that this obstructing type of colostomy may be a contributory cause, and for this reason we have made it our practice for the last two years

to open every colostomy on the table at the conclusion of the operation, and so far no case of paralytic ileus has followed this procedure

Mechanical Obstruction.—This has accounted for 5 post-operative deaths, in 1 case a band was found post mortem obstructing the transverse colon, and in 2 cases the small intestine was proved to have become adherent and kinked, once at a transverse colostomy and once to the dropped-back colon in one of the early cases in this series (1913) where the bowel had been divided and a terminal colostomy established

In 2 cases a knuckle of small intestine became strangulated through the foramen on the lateral aspect of a rectus colostomy

Case 8—G G, a man, aged 53, with an operable anal carcinoma. A left rectus colostomy and perineal excision of the rectum was carried out in one stage. On the third post-operative day he became distended and began to vomit small intestinal contents, laparotomy revealed a small-gut obstruction due to a knuckle of the bowel entering from above downwards into the foramen on the outer side of the colostomy, the small intestine beyond this point was collapsed. The bowel was reduced, but the patient died four hours later (*Fig 365*)

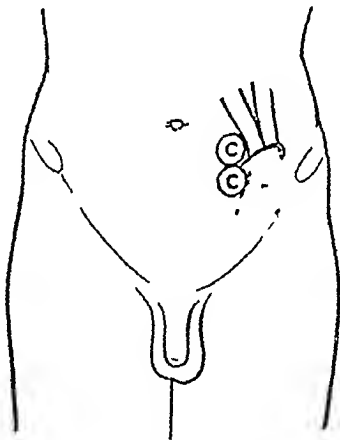


FIG 365—Case 8

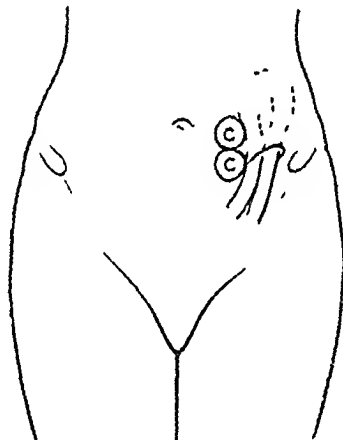


FIG 366—Case 9

Case 9—Mrs E S, aged 55, with an operable carcinoma in the ampulla of the rectum. A left rectus colostomy was established, and fourteen days later the rectum was excised by the perineal route. There was some post-operative vomiting and late on the second day after the excision operation the abdomen became distended and the patient rapidly failed. Post-mortem examination showed a small-gut obstruction 4 ft from the duodenojejunal flexure, from below upwards round the colostomy (*Fig 366*)

Since these cases were reported, together with 3 others in 1928 (Gabriel¹), the expedient of closing the lateral space by suture when doing a colostomy has been generally adopted, and no further example of this type of obstruction has occurred in the immediate post-operative period in St Mark's Hospital

Toxæmia from Pre-operative Obstruction—The added risks of colostomy when carried out in the late stages of obstruction are indicated by the cases in this series. 22 patients were admitted with marked abdominal distension, and of these 7 died after operation (32 per cent). Four of these died from toxæmia at an average of three days after operation, the remaining 3 deaths falling under other headings (pulmonary œdema, paralytic obstruction, and prolapse of the small intestine)

Prolapse of Small Intestine.—This complication has accounted for 9 fatalities in this series, and on analysis two points are at once apparent in regard to (1) The incision used for the colostomy, and (2) The age of the patient

1 In every case the colostomy was done through a left rectus incision, and the small intestine subsequently made its way through the upper and inner aspect of the wound. The increased risk of a rectus incision with direct underlying pressure from the small intestine is further shown by the other cases with this complication who recovered after operative reduction of the prolapsed bowel or omentum—namely, 3 cases of small intestinal prolapse (2 rectus colostomies, 1 left oblique), and 3 cases of prolapse of the great omentum (all rectus colostomies). There are thus altogether 15 cases of prolapse of the small intestine or omentum, of which only 1 was through a left oblique incision. The high mortality attached to prolapse of the small intestine is shown by the death-rate of 75 per cent (9 deaths out of 12)

2 That advanced age predisposes to this complication is shown by the fact that in 7 patients the age was between 60 and 77 years, with an average age of 67. In these cases the prolapse occurred as a rule between the ninth and the fifteenth day after operation. Post-operative bronchitis with continual coughing is an evident contributory cause of a prolapse, and so also is the lack of healing capacity, which would be expected in advanced cancer cases. This is shown by the fact that in 3 cases marked ascites was found at operation and 1 patient had both free fluid and advanced intestinal obstruction.

In 2 cases the patients were much younger, a female of 32, and a male of 46. In these cases the prolapse occurred through a left rectus colostomy incision on the first and second days respectively after operation, and were due presumably to inadequate closure of the abdominal wall round the emerging colon.

In all cases the prolapsed bowel was carefully cleaned and replaced into the abdominal cavity under gas and oxygen, spinal, or local anaesthesia, and the abdominal wall was re-sutured round the colostomy. The 9 subsequent deaths took place within five days from shock or sepsis (6 in number), or much later between the eighth and twenty-second day (3 in number), being then considered to be due to asthenia or bronchopneumonia.

Uræmia—Urinary failure is considered to have been the cause of 6 deaths in this series. In 1 case multiple abscesses were found post mortem in both kidneys, due to obstruction of the ureters by growth; 1 man had retention of urine due to urethral strictures of long standing; 1 man died in uræmic coma, and the remaining 3 had clinical or post-mortem signs of pyelonephritis.

Malignant Cachexia—In 5 cases patients with large inoperable carcinomata went gradually downhill after colostomy, and died at an average of twenty-one days after operation.

Miscellaneous Complications—In this group there are 3 deaths the cause of which cannot be ascertained with accuracy, 1 death from diabetic coma, and 1 death from continued hæmorrhage from the proximal colostomy opening. This occurred in a man of 70 with an inoperable growth and secondary deposits in the liver. The bleeding from the bowel began on the thirteenth post-operative day and continued till his death three days later. It may have arisen in a malignant ulceration, or a polyp higher up the colon, or possibly from a duodenal ulcer.

DURATION OF LIFE AFTER COLOSTOMY

An inquiry into the after-histories of patients treated by palliative colostomy has shown certain facts which are set out in *Table III*. The subsequent duration of life is shown exactly in months and years according to the stage of the disease at the time of operation, a method which will, we think, be more useful than striking an average for the entire series. The following points are noteworthy —

Operable Cases.—The 10 operable cases treated by colostomy alone were chiefly ones who refused to submit to perineal excision of the rectum after colostomy had been established. As would be expected, these patients lived for fairly long periods, half the number living for more than 3 years after operation. One patient (a man aged 66 at operation) lived for 6 years and 11 months, and one (a man aged 69 at operation) is still alive 5½ years afterwards, in this case a very large fixed neoplasm is now present in the rectum, which a biopsy has proved to be an adenocarcinoma*.

Table III—DURATION OF LIFE AFTER RECOVERY FROM PALLIATIVE COLOSTOMY (310 CASES)

	DIED IN LESS THAN 3 MONTHS AFTER OPERATION	DIED BETWEEN 3 AND 6 MONTHS AFTER OPERATION	DIED BETWEEN 6 AND 12 MONTHS AFTER OPERATION	SURVIVED 1-2 YEARS	SURVIVED 2-3 YEARS	SURVIVED 3-4 YEARS	SURVIVED 4-5 YEARS	SURVIVED MORE THAN 5 YEARS
Operable cases treated by colostomy alone	—	1 (cardiac failure)	—	2	2	2	1 (living)	2
Inoperable cases	30	33	65	71 (including 13 living)	24 (including 2 living)	11 (including 1 living)	3	1
Inoperable cases with secondary deposits in liver or peritoneum	15	21	17	4	4	—	1	—

NOTE.—Cases now living at a period of less than 12 months after operation are excluded from this table.

Inoperable Cases—In this group, totalling 238, the growths were inoperable as a result of local extent and fixation to surrounding structures. *Table III* shows that rather more than 50 per cent died within twelve months after operation. A large group, 30 per cent, survived for between 1 and 2 years, 10 per cent reached the 2- to 3-year period, and 15 cases (6 per cent) survived for 3 years and more. Only one case lived for more than 5 years, this was a man aged 53 for whom a colostomy was done at a stage when he had a circular constricting growth in the rectum adherent to the base of the bladder. Four and a half years later he was re-examined, in spite of some pelvic pain he was able to do half-time work at a railway station, and his colostomy was acting well. He died 5 years and 8 months after the original operation.

* This patient has recently died, exactly 6 years after the colostomy operation.

Cases surviving for as long as this are rare, but there is a certain type of inoperable rectal carcinoma which shows little tendency to lymphatic or venous spread. After a colostomy has been established the growth remains localized in the pelvis and slowly extends until a very large fixed mass results. Pressure symptoms are often late in becoming manifest and the patient is enabled to work and be a useful member of society until an advanced stage of the disease is reached. In cases such as this a colostomy is indeed a very valuable measure for palliation.

Inoperable Cases with Metastases in the Liver or Peritoneum—The short expectation of life in these advanced cases is indicated by the 62 cases shown in the table. More than 50 per cent died within 6 months, and 85 per cent were dead in less than a year after operation. Five cases, however, are known to have lived for more than two years after colostomy.

Case 10—Male, aged 36, with a large inoperable rectal carcinoma, peritoneal secondaries found at operation, colostomy. Lived for 2 years and 1 month.

Case 11—Male, aged 30, inoperable rectal carcinoma filling the pelvis. Secondary deposits found in the great omentum, which on section were proved to be colloid carcinoma. Lived for 2 years and 4 months after colostomy.

Case 12—Male, aged 64, inoperable growth in rectum with a secondary nodule felt in liver at operation. Lived for about 2½ years after colostomy. Cuprase injections given after operation.

Case 13—Female, aged 60, small mobile rectal growth proved by section to be a carcinoma. At operation the liver was found to be studded with numerous nodules which were considered to be secondary deposits, probably the result of portal vein embolism. This patient lived for 2½ years after colostomy, cuprase injections were also given after operation.

Case 14—Male, aged 62, with an inoperable rectal growth. At operation enlarged glands were found extending high up at the root of the pelvic mesocolon, and secondary deposits were present on the peritoneum. Lived for 4 years and 2 months after colostomy.

Results such as these indicate the difficulty of making a prognosis in advanced rectal cancer. In 4 of these cases where the rectal growth was very extensive, we think the unexpected prolongation of life must have been brought about principally by the colostomy with relief of intestinal obstruction and toxæmia, and the ability to take a full diet afterwards must have aided the patients' resistance to the cancer.

Age and Sex—We have inquired particularly into the results of colostomy when done for patients aged 70 or over, to see if there is sufficient justification for operation at this advanced age. In 66 patients over 70 years of age there were 13 operative deaths (*see Table I*). Many of the early cases in the series are untraced, but the after-histories of 41 (34 males, 7 females) are known. Seven patients (6 males, 1 female) are known to have lived for 2 years or more, the maximum length of life being 3½ years (2 male cases). The majority lived for shorter periods, and we find that the average post-operative duration of life in the 41 survivals from operation is 13 months. These facts show that colostomy can be a satisfactory operation in advanced age, particularly in males, for the operation mortality in females at this age is high, and we think that old men are, on the whole, the better subjects for the operation, both mentally and physically.

A comparison of the sexes may also be made as regards the likelihood of survival for three years or more. *Table III* shows 21 patients in this group, of whom 18 were males, and 3 females, after making due allowance for the 3:1 proportion of males to females in this series, this finding suggests that cancer of the rectum runs a more chronic course in males than females.

LATE COMPLICATIONS AFTER COLOSTOMY

These complications have been studied both in respect of the preceding cases of palliative colostomy, and also in a series of 370 cases of recovery after perineal excision with permanent colostomies. With regard to the function of the colostomies, we find that in 86 per cent the function has been satisfactory. Certain cases have been followed up over a long course of years, and there is no doubt that with a regular routine and careful management colostomies can give good service with very little disability. Certain of the complications which will now be described have occurred to a minor degree in some of these cases, but have not disturbed the satisfactory function of the colostomies. The following complications are known to have occurred: (1) Stenosis, (2) Ventral hernia, (3) Spur retraction, (4) Prolapse, (5) Fistula into the colon.

Stenosis—This is the most frequent complication and is due to the development of a contraction ring of fibrous tissue at the junction of the skin and mucous membrane at the colostomy. The abdominal wall musculature plays no part in it. Skin stenosis usually occurs quite early, and in some cases we have known this to happen within six weeks after the colostomy operation, moreover, it may be a very troublesome condition, which in some cases has shown a persistent tendency to recur, possibly owing to a tendency to epithelial overgrowth.

A common sequel to skin stenosis of a colostomy is a subcutaneous bulging of the colon. In these cases the colon appears to expand for a considerable extent into the subcutaneous tissues superficial to the external oblique or rectus, and the condition is due to the action of intracolonic pressure when the colostomy opening is stenosing. This bulging is most marked in obese subjects and is rarely seen in thin patients, in whom there is little subcutaneous tissue.

Treatment in early cases consists in daily, later weekly, dilatation with the finger, if this is done sufficiently early, while the scar is still soft, the contraction may be overcome. In late cases, and those in which digital dilatation has failed a secondary operation must be done with a view to excision of the scar and an ellipse of skin round the colostomy. In addition, the actual orifice of the colostomy usually presents as a fibrous ring of inadequate size, which needs to be enlarged by incision. When a subcutaneous bulge is also present some of the overlying skin must be cut away, followed by re-suture of the mucous membrane to the new skin edge.

We have notes of 41 cases of skin stenosis, of which slightly more than 50 per cent needed operative treatment.

Ventral Hernia (*Fig 367*)—This has occurred in a small number of cases, not exceeding 10 per cent of the total, with a slightly greater incidence in left iliac colostomies compared with those brought out through the left rectus. The largest herniæ which we have seen have been in cases in which (1) Owing to misjudgement of the incision the colostomy has been made at the outer border of

the rectus, i e , neither a true rectus nor a true iliac colostomy , and (2) Those in which a cup variety of belt has been worn by the patient (*Fig 368*) In every case the bringing out of a colostomy must have some tendency to weaken the



FIG 367 —Ventral hernia

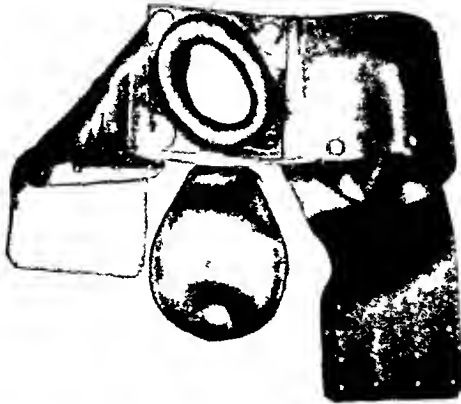


FIG 368 —Cup belt and bag

abdominal wall, and for this reason it should be one's aim to obtain sound primary healing of the wound, and to maintain firm support during the months immediately following operation The wearing of the large celluloid cups which are still all too often supplied to patients by surgical instrument makers is almost certainly

followed by the formation of a large ventral hernia, which in time seriously interferes with the colostomy function. This complication is one which it is eminently better to prevent rather than to cure, and this can be done chiefly by careful operative technique, by aiming at subsequent asepsis, and the wearing of a flat supporting belt. Only in rare cases is it necessary to perform a reparative operation.

Spur Retraction—This is an important complication which results in complete dysfunction of the colostomy, with passage of faeces into the distal colon, and is often found in conjunction with stenosis, subcutaneous bulging, and ventral hernia. Retraction occurs most commonly in cases in which owing to shortness of the mesocolon, or to excessive depth of fat in the abdominal wall, it has proved difficult to bring the colon to the surface. Undue tension subsequently takes place in the bowel, and there may be a persistent tendency to retraction. Various devices may need adoption to deal with the difficulty and prevent the occurrence of this complication. (1) At operation thorough mobilization of the colon by incision of adventitious peritoneal folds usually found on the outer side of the mesocolon, (2) Excision of wedges of subcutaneous fat round the incision, or, rarely (3) Abandoning the plan of making an iliac or pelvic colostomy in favour of a transverse colostomy through a new incision. If a 'rod' colostomy is made, the rod should be left *in situ* for at least fourteen days in order to get the colon well adherent above the abdominal wall, alternatively, a 'bridge' colostomy may be done by bringing a strip of skin or aponeurosis (rectus sheath or external oblique) through a hole in the mesocolon. It should be noted, however, that if a skin bridge is used, there appears to be some additional risk of late skin stenosis, to avoid which excision of skin round the colostomy is necessary.



Fig 369—Prolapse

some additional risk of late skin stenosis, to avoid which excision of skin round the colostomy is necessary.

The treatment of a colostomy with a retracted spur must be operative and consists in dissecting out the retracted bowel and refixing by one of the methods mentioned above.

Prolapse (Fig 369)—This is a rare complication, and we have notes of only 12 cases in this series. As in the case of rectal prolapse, a colostomy prolapse may be complete (entire thickness of colon) or incomplete (mucous membrane only), it usually takes place from the upper opening, or occasionally from the lower or both, openings. Prolapse is always associated with a patulous opening in the abdominal wall. Treatment in slight cases is by firm support, and in severe cases when the patient is much troubled by mucoid discharge, amputation of the prolapse becomes necessary and can be effected with only slight risk.

Fistula into the Colon—This we have known to occur in 2 cases, it has probably been due to ligation of appendices epiploicæ which have happened to contain diverticula. This complication can be prevented by leaving untouched any thick appendices which are found arising from the colon near the skin level, instead of excising them for neatness it is preferable to leave them or tuck them back beneath the skin level.

TECHNIQUE OF COLOSTOMY

Preparation—Unless the patient is definitely obstructed, a palliative colostomy is not to be regarded as an emergency operation. A slight degree of abdominal distension will in most cases subside if the patient is put to bed on a fluid diet, with small repeated doses of liquid paraffin and saline aperients. glycerin enemata should be given if necessary. The mouth should be cleaned up, the urea excretion should be estimated, and steps taken to improve it if possible. The condition of the heart and lungs must be estimated, and thought given to the most suitable type of anæsthesia. With rest and careful preparation on these lines we believe that much can be done to lower the operative mortality in these cases.

Incision and Fixation of the Colon—The tendency at St Mark's Hospital in recent years has been to perform an increasing proportion of colostomies through a left oblique muscle-splitting incision. The advantages of this incision are —

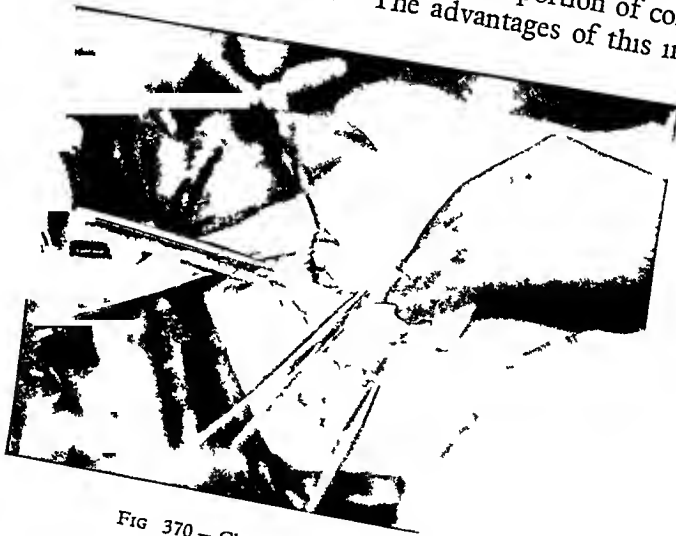


FIG 370—Closure of the lateral space

- 1 It is placed directly over the iliac colon, which can usually be brought comfortably to the surface, leaving the pelvic colon undisturbed should any further operative procedure be planned from below.
- 2 The lateral space between the emerging colon and the parietal abdominal wall can be readily closed by suture, thus preventing the possibility of small-intestinal strangulation here.
- 3 The figures already adduced show that prolapse of the small intestine is rare through an oblique incision when compared with a rectus or paramedian incision.

Against this it must be admitted that a left rectus incision has some advantages in that the colostomy is placed in a slightly better position for the patient's management, and there may be less likelihood of a subsequent incisional hernia, but after assessing these various points we believe that a left oblique colostomy, if done with attention to the following points, is the safest and most satisfactory method, particularly when done in conjunction with a careful abdominal exploration through a right paramedian subumbilical incision.

A 'gridiron' incision, 4 in long, is made in the left iliac region, occasionally it may be necessary to incise the internal oblique for an inch transversely to its fibres at the medial end of the incision. The iliac colon is found immediately deep to this incision and is brought out, being held gently in a wet pack. On doing this a peritoneal fold is produced running outwards and forwards from the lateral aspect of the bowel to the cut edge of the parietal peritoneum. A No 0 catgut suture on a curved atraumatic needle is passed from within outwards along the free edge of this fold, and when tied effectively closes what would otherwise become an artificial foramen (*Fig 370*). A glass rod, $\frac{1}{4}$ in in diameter, and $3\frac{1}{2}$ to 4 in in length, with smooth rounded ends, is now passed through the mesocolon immediately above the stitch along the lateral fold. Injury to the veins can usually be avoided by selecting an avascular part and gently pressing into it one end of the rod, while an assistant with a pair of non-toothed dissecting forceps scratches on to the rod as it is made to present on the opposite side. The rod is prevented from slipping out by placing on each end a separate piece of rubber tubing (A half circle of tubing is cumbersome and does not seem to have any additional advantage).



FIG 371—Finished colostomy

Closure of the Wound—The peritoneum is closed above and below the bowel with No 1 chromic catgut, leaving a space which will take both limbs of the colostomy without undue compression. In practice it is found advisable not to close the peritoneum so tightly that a finger cannot fairly be passed in alongside the bowel. The internal oblique is sutured if necessary, and the external oblique is repaired with interrupted catgut sutures, each of which takes up a small portion of the underlying muscle. We attach some importance to this point, for it ensures closure of an intermuscular space into which the colon might subsequently bulge. Care must be taken not to suture the external oblique too closely to the bowel, and in addition it is often advisable to make

short relieving incisions, with blunt-pointed scissors, into the sharp edges of the external oblique aponeurosis. These incisions are made at right angles to the fibres opposite the centre of the space left for the emerging bowel. An ellipse of skin is excised from both margins of the wound immediately opposite the colostomy

to avoid the common late complication of skin stenosis. The remainder of the wound is closed with interrupted silkworm-gut sutures, which include the external oblique and thus obliterate a potential subcutaneous space into which the bowel at a later date has not infrequently become herniated (*Fig 371*)

Ligation of Appendices Epiploicæ.—Redundant appendices epiploicæ are now ligated and excised, this should be done principally in relation to the prominent part of the colon, with care not to ligate any appendices which arise close to the skin level



FIG 372—Elastoplast dressing

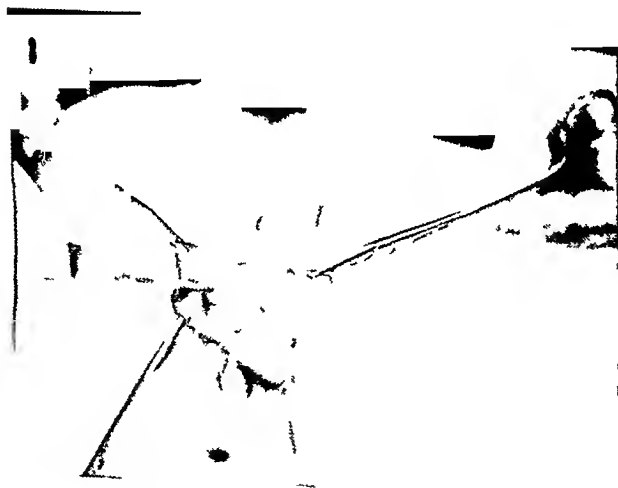


FIG 373—Opening the colostomy with a cautery

Dressings and Immediate Opening of the Colostomy.—If a paramedian exploratory incision has been made, it is dressed with gauze and sealed with elastoplast. The colostomy incision is dressed similarly, and after carefully tucking flat pieces of dressing gauze under the rod, a supporting transverse strip of elastoplast

is placed each side (*Fig 372*) The wound is additionally protected by sterile towels and packs, and with a Paquelin or Post cautery the colostomy is now opened widely by transverse and longitudinal incisions (*Fig 373*) Any solid faecal masses which present are removed The immediate introduction of about 5 oz of warmed olive oil into the proximal limb of the colostomy has, we think, proved beneficial to patients (*Fig 374*) The oil can be run in easily through a No 16



FIG 374—Introduction of olive oil

rubber catheter, it is always retained and greatly assists in lubricating the bowel and allowing the easy passage of flatus Finally a dressing of dry gauze and wool is applied

Subsequent Care—The patient is kept for the next week on a low-residue, chiefly fluid diet As a rule colostomy wash-outs are inadvisable before removal of the sutures, and we think that most patients run a quieter and more comfortable post-operative course if wash-outs into the proximal bowel are not given Small doses of liquid paraffin may be given on the third day after operation, and usually a colostomy action begins on the fourth day

Flatus is passed per colostomy from the time of operation, and the absence of post-operative pain from gas distension of the colon is very marked in those cases in which immediate opening of the colostomy has been done The Ward Sisters in charge of large numbers of colostomy cases are in a good position to give an opinion on this matter, and we find among them a general agreement as to the almost complete absence of post-operative pain after laparotomy and colostomy with immediate opening of the bowel The elastoplast is removed on the third or fourth day and the underlying gauze changed primary healing has been obtained in practically all cases treated by this method Sutures are removed from the colostomy incision about the sixth or seventh day The glass rod is left *in situ*

for fourteen days and may then, with advantage, be replaced by a rubber tube with over-turned ends to prevent it slipping out (*Fig 375*), this tube can be left for another one or two weeks before final removal, and helps to maintain a good spur until the bowel is firmly adherent

With regard to the subsequent management of the colostomy there are these two alternatives (1) The training of the bowel by careful dieting and regular meals until the habit of regular colostomy actions once or twice a day has been established, and (2) The giving of a soap and water wash-out into the proximal colon at a regular time each day, morning or evening This routine has some



FIG 375 —Rubber tube substituted for glass rod

advantages, especially for manual workers, and in many of these cases has been satisfactory over a long period of years There are, however, some patients who after continued wash-outs develop a chronic catarrh of the colon, with excessive formation of mucus and sometimes irregular or incomplete return of the daily wash-out At the same time there is a loss of the natural dilating effect of formed faeces upon the colostomy opening For these reasons we think that after colostomy a patient should be encouraged first to try and obtain natural colostomy actions, if this fails for reason of loose or irregular actions, then the daily wash-outs must be started

In very infirm subjects and in those lacking responsible relatives or nursing facilities at home, the arrangements for the patient's subsequent after-care need to be carefully considered, and in the poorer classes permanent institutional treatment is often desirable after colostomy

SUMMARY

- 1 The value of palliative colostomy for rectal carcinoma and the operative mortality is discussed
- 2 The causes of death have been analysed and the duration of subsequent life has been shown, together with the late complications
- 3 An operative technique for colostomy has been described, an important

feature being the immediate opening of the bowel. This is applicable both to obstructed and non-obstructed cases, it has already considerably reduced the mortality rate and will, we think, prevent many of the late complications.*

The statistical information regarding these cases has been obtained from the Cancer Follow-up Scheme of St Mark's Hospital, which has been financed by the Medical Research Council since 1922. We are indebted to Mr H J R Bussey, B Sc, of St Mark's Hospital, for the photographs illustrating this article.

REFERENCE

¹ GABRIEL, W B, *Proc Roy Soc Med*, 1928, **XXI**, 1433

* In 41 operations with this technique for inoperable rectal cancer there have been 2 deaths (5 per cent). In a further 23 operable cases there have been no deaths attributable to the colostomy.

ADENOMATA OF THE PITUITARY, WITH SPECIAL
REFERENCE TO PITUITARY BASOPHILISM OF CUSHING

BY WILLIAM SUSMAN

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In the past, benign tumours of the anterior pituitary have been considered important because in some cases they gave rise to acromegaly, and in others to disturbances chiefly due to mechanical reasons. In recent years, however, Cushing¹ has described a clinical syndrome which he has termed 'pituitary basophilism', and which he considers to be due to hypersecretion arising from a basophilic adenoma or basophilic hyperplastic area in the anterior pituitary. Cases showing this syndrome have a rapidly acquired and painful adiposity, lumbospinal pains, sexual dystrophy, hypertrichosis, and a dusky skin.

If the syndrome described by Cushing is due to the hypersecretion of the basophilic cells in an adenoma or in a hyperplastic area in the anterior pituitary, then such lesions must be closely associated with cases showing this syndrome. However, if tumours of this type are common, then they are of no etiological significance, and their presence in these cases is only due to chance.

In the endocrine glands the whole question of benign tumours is intimately bound up with the process of hyperplasia in its general sense, in other words, many, if not the majority, of such growths are in all probability not neoplasms in the true sense. If these benign tumours are hyperplastic rather than neoplastic, then, as tissue damage and repair are usually associated with hyperplasia, such changes should be present in any series including a considerable number of pituitaries. An investigation of a series of 260 hypophyses obtained from routine post-mortem examinations showed that the hypophysis in this respect did not differ from the remainder of the ductless glands. In short, as the hypophysis is damaged quite frequently, repair in that organ is not uncommon, and therefore it is not in the least surprising that so-called adenomata or hyperplastic areas should occur.

The series forming the basis of this investigation is made up of 260 cases examined at the Manchester Royal Infirmary in the course of the routine necropsy examinations. The gland was examined in all cases in which the cranial vault was opened. The only limiting factors were time and the condition of the tissues. The hypophysis of each case was cut antero-posteriorly through the infundibulum and fixed in Helly's fluid. Each half of the gland was prepared for paraffin sections, and those sections from the infundibulum region were stained by hæmalum and eosin. When in doubt, acid fuchsin-methyl green or Weigert's iron-hæmatoxylin and eosin methods were utilized. Both of these latter methods differentiate the cells of the anterior pituitary very well. On the whole, it must be considered that the infundibulum region alone was examined, although a few of the glands were examined in serial sections.

Of the 260 glands in this series, 22 contained 23 tumours, and, of these, 20 were of a purely incidental character and had given rise to no symptoms. The

histories from these tumour cases were quite typical of the disease evident at the time the patient was admitted into hospital, and at the time of death. The cases in which the tumours had given rise to symptoms were (1) A case of acromegaly in which the anterior pituitary contained an acidophilic adenoma (6×8.5 mm), and (2) A case in which an acidophilic adenoma 25 mm in diameter had given rise to pressure symptoms. Thus benign tumours of an incidental character occurred in about 8 per cent of the cases of this series.

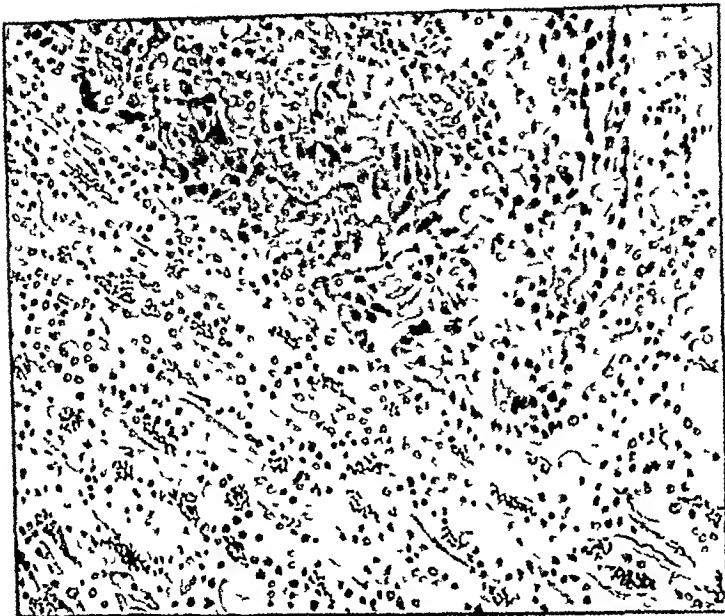
The incidental tumours varied from 0.2 mm in diameter to 7×3.5 mm. Of the acidophilic type there were 4 (16 per cent), of the basophilic 8 (3 per cent), and of the chromophobe 5 (2 per cent). The remainder formed a miscellaneous group, in one case the tumour was made up of columnar cells, while in 3 others small tumours consisting of greyish cells of a rather indifferent appearance were present in the infundibulum and in the pars nervosa. The full details are shown in Table I.

Table I—PITUITARY ADENOMATA, 23 CASES (2 IN 1 CASE)

Basophilic —		SIZE IN MM
M 49	Pernicious anemia	1.5
F 26	Acute encephalitis	1×0.5
F 57	Otitis media	0.3
F 40	Pellagra and confusion (Fig. 376)	0.3
M 52	Acute mastoiditis (Fig. 377)	1.5×2.3
F 22	Diabetes mellitus	1.5
M 36	Addison's disease (tuberculosis)	0.2
M 31	Glioma	5×2
Acidophilic —		
M 58	Shock following fall	0.3
F 24	Pituitary adenoma	25.0
M 17	Renal infantilism	2.0
M 26	Fracture skull, meningioma dura	0.2
F 62	Pellagra and acute pancreatitis	7×3.5
F 66	Acromegaly	6×8.5
Chromophobe —		
M 25	Glioma and military tuberculosis	1.0
M 42	Cerebral hæmorrhage	1.5
F 44	Addison's disease	1.8
M 42	Tabes	0.4
M 55	Cerebral hæmorrhage	0.2
Miscellaneous —		
F 55	Cerebral hæmorrhage, columnar-celled	0.6
M 48	Cerebral hæmorrhage (in infundibulum)	0.3
F 37	Diabetes mellitus (pars nervosa)	1.5
M 50	Ruptured cerebral aneurysm (in infundibulum)	0.3

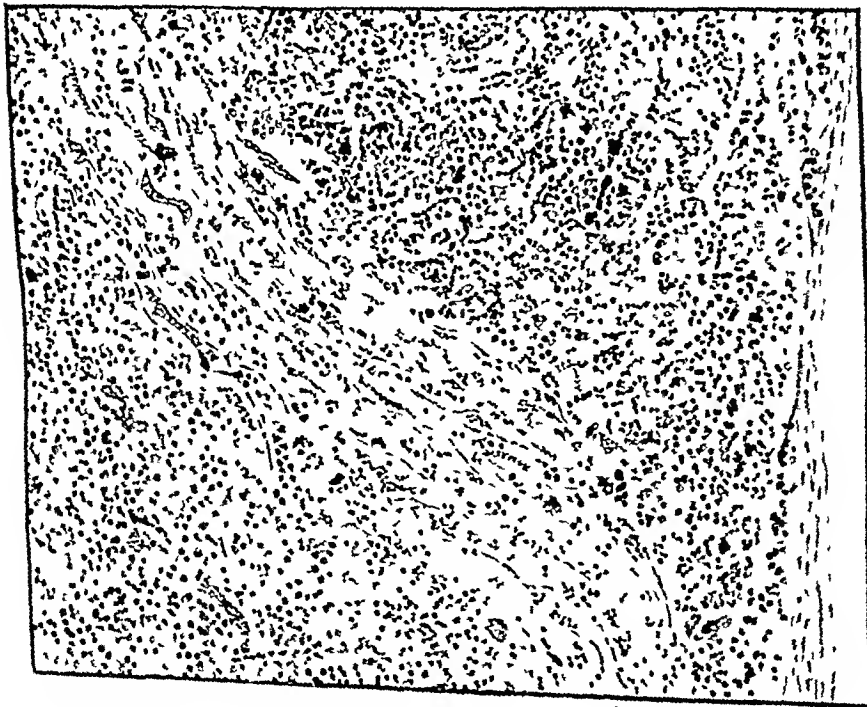
In Table II the age and sex incidence of this group of tumour cases is compared with those of the whole pituitary series.

The available data show, therefore, that incidental tumours occurred in 8 per cent of the cases in this series when the gland was examined in the infundibular region only. Had the glands been further examined, then most probably the incidence would have been proportionately higher. Further, the basophilic tumour was the commoner type present. The age and sex distribution were roughly comparable with those of the general series. Nor did the diseases prevalent in these



D DAVISON,

FIG 376—Case 1 Female, aged 40 Pellagra The tumour is about 0.4 × 0.6 mm and consists of basophilic cells which are about normal in size The growth is well demarcated from the glandular tissues (Stained by hæmalum and eosin)



D DAVISON

FIG 377—Case 2 Male, aged 52 Acute mastoiditis The tumour is 1.5/2.3 mm and is made up of basophilic cells of the small hyperplastic type The nodule is quite distinct from the surrounding glandular tissue (Stained by hæmalum and eosin)

cases give any specific indication, but, on the contrary, they varied greatly. Pellagra was present in 2 cases, cerebral hæmorrhage in 4, and tumours in 4, while the remaining cases were each of a different type. All had histories typical of the disease indicated for each case. These tumours therefore occurred in no specific age, sex, or disease group. This applied equally well for each of the several types of incidental adenomata of the anterior pituitary.

Table II—AGE AND SEX INCIDENCE OF PITUITARY TUMOURS

AGE GROUP	ADENOMA SERIES				GENERAL PITUITARY SERIES		
	M	F	Total	Per Cent	Number	Per Cent	Per Cent of Adenoma Cases in each Age Group
0-9	—	—	—	—	16	7.6	0
10-19	1	0	1	4	25	10.0	4
20-29	2	3	5	23	38	15.2	13
30-39	2	1	3	14	34	13.6	9
40-49	4	2	6	27	52	20.8	12
50-59	5	0	5	23	47	16.8	11
60-69	0	2	2	9	25	10.0	8
70 +	—	—	—	—	9	3.6	0
Totals	14	8	22	100	246*	—	—

* Males 151, females 95

Then in what way do these tumours differ from the actively secreting tumours of the acromegalic? The size of the tumour is no certain indication, as can be seen from *Table I*. In the acromegalic tumour the cells are hypertrophied (*Fig 378*). In contrast, the cells of the incidental tumours (*Figs 379, 380*) and of the tumour which gave rise to pressure symptoms (*Fig 381*) were hyperplastic, and from a functional standpoint these cells were probably ineffective. This one may reasonably infer from what occurs in the thyroid.

Table III—BASOPHILIC CELLS HIGH IN 21 CASES *

AGE GROUP	NO OF CASES
0-9	2
10-19	1
20-29	3
30-39	0
40-49	6
50-59	6
60-69	1
70 +	1

* Males, 16, females, 5. Age in 1 case not available

But even if the basophilic adenomata had produced an effective and distinctive secretion, what type of symptoms might one expect? The whole series of 260 cases included 21 cases in which the basophilic cell content of the anterior pituitary was definitely high. A study of these cases showed that the age and sex incidences were those of a fair sample (*Table III*). Acute diseases accounted for 5 cases, tumours for 5, chronic nephritis for 2, and epilepsy for 2. The remaining cases were each of a different type. Thus, an abundance of basophilic cells in the

ADENOMATA OF THE PITUITARY

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FIG 378—A, The acidophilic adenoma in an acromegalic hypophysis. The cells are larger than those in the other types shown in Figs 379-381. B, The neighbouring anterior pituitary tissue ($\times 130$)

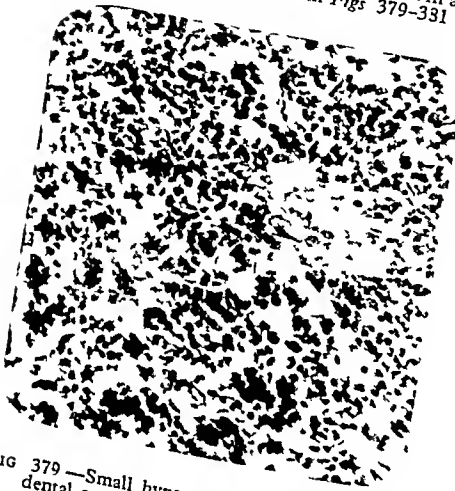


FIG 379—Small hyperplastic cells in an incidental acidophilic adenoma ($\times 130$)

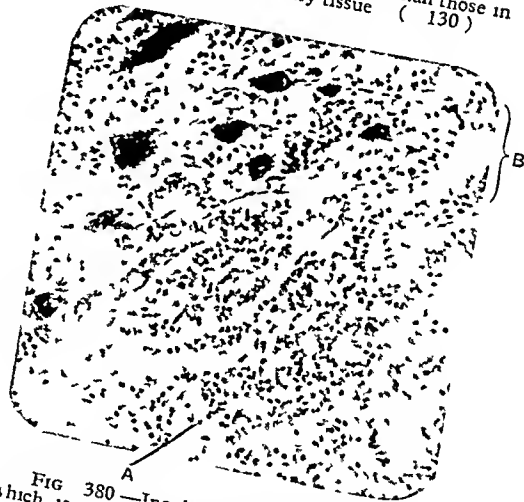


FIG 380—Incidental basophilic adenoma which is made up of small hyperplastic cells. A Tumour, B, Anterior pituitary ($\times 130$)

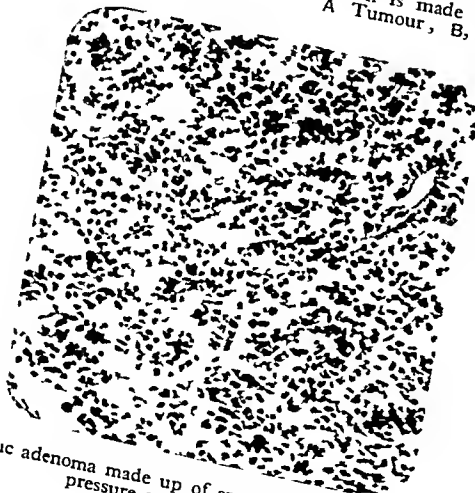


FIG 381—An acidophilic adenoma made up of small hyperplastic cells, it had given rise to pressure symptoms ($\times 130$)

anterior pituitary is not peculiar to any age, sex, or disease group. If these cells produced a hormone important to the sex mechanism of the individual, a definite difference could be expected, both with justification and without exception, as between infants and children as sexually immature and practically sexless on the one hand, and the adults on the other. This, however, was not the case, as the figures in the tables have indicated. The incidence of cases showing a high basophilic cell content in the anterior pituitary was high between the ages of 40 and 60 years, but almost 40 per cent of all the cases in the whole series belonged to this age period.

The conclusion is, therefore, that the histological data from this series of 260 hypophyses give no support to the contentions either of Zondek,² that the basophilic cells of the anterior pituitary secrete a sex hormone, or of Cushing, that hypersecretion of basophilic cells caused by the presence in the anterior pituitary of a basophilic adenoma or of an area of basophilic hyperplasia will give rise to the syndrome which he has termed 'pituitary basophilism'. Adenomata as a whole occur in 8 per cent of cases, the basophilic type in 3 per cent, and they are therefore too common to be of any special significance.

I wish to thank Dr Tattersall, of the County Mental Hospital, Prestwich, for the acromegalic hypophysis, and Mr H. C. Taylor for his care in preparing the photomicrographs.

REFERENCES

¹ CUSHING, H., *Bull. Johns Hopkins Hosp.*, 1932, 1, 137.

² ZONDEK, B., *Klin. Woch.*, 1933, vii, 1.

PERFORATION OF CARCINOMA OF THE STOMACH INTO THE GENERAL PERITONEAL CAVITY

BY IAN AIRD

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A HUNDRED and ten years ago Laennec noted in his case book, "*Ventriculus hodie ruptus est in loco scirrhi et jam peritonitis adest et tympanites peritonealis*",⁴⁰ and few since have been fortunate enough to make such a diagnosis with the same confidence and the same accuracy. Still to-day no clear clinical picture assures for us the pre-operative recognition of perforated carcinoma of the stomach, the infrequency of this catastrophe in even the widest surgical practice and the lack of a comprehensive English description of the condition conspire to render exact diagnosis uncertain. A study of the details of available cases is thus justified, and the rarity of a correct diagnosis—definitely claimed in only five reported cases—renders such a study not only justified but imperative. In the consideration of perforated carcinoma of the stomach which follows, one case of the writer's will be described, 7 other cases treated in the Royal Infirmary, Edinburgh, during the last ten years will be summarized, and with these will be examined 71 cases collected from the available literature.

CASE REPORTS

Case 1 (author's case)—A ship's mate, aged 27, was seized while out walking by epigastric pain, slight at first, but increasing gradually to become intense after an hour, and generalized then over the whole abdomen. Vomiting of yellow fluid was frequent thereafter, but failed to relieve the pain. Slight irregular epigastric pain, not consistently related to food, had been present untreated for some years. This pain had been more severe, and almost continuous for a few weeks before admission to hospital. No vomiting or melaena had occurred, and there had been no loss in weight.

ON ADMISSION—On admission to hospital, six hours after the onset of the attack, the pain eased and the vomiting ceased. The temperature was 97.6°, pulse 120, regular, and soft, respirations 22. Tenderness and rigidity were intense and generalized, but maximal in the epigastrium. No distension was present, a palpable tumour was not felt, and there was no dullness in the flanks. Liver dullness was diminished.

OPERATION—A diagnosis of perforated peptic ulcer having been made, the abdomen was opened by a right paramedian rectus-displacing incision, under gas and oxygen anaesthesia. The peritoneal cavity was evacuated of gas and of about one pint of serofibrinous straw-coloured exudate. The parietal and visceral peritoneum was everywhere congested. No perforation was found on the anterior wall of the stomach or duodenum, but the omental bursa having been opened through the transverse mesocolon, a perforation was found on the posterior wall of the stomach, 1 in. in diameter, and situated near the lesser curvature about 2 in. from the pylorus. The edges of the opening were ragged, white, and crumbling, and the perforation had occurred through the posterior flap of a saddle-shaped ulcer which involved the lesser curvature and the greater part of the girth of the stomach. No suspicion arose that this ulcer might be malignant, until a mass of large hard glands was observed in the lesser omentum. One of these was excised. An attempt was now made to close the perforation—a matter of extreme difficulty on account of the friability of the diseased stomach wall around the opening.

Gastrectomy seemed at one point the only solution, but ultimately a closure was effected by mattress sutures, and the closed perforation was overlaid by transverse mesocolon. A suprapubic stab incision was made for the transmission of a drain to the rectovesical pouch, and the parameian wound was closed in layers. Section of the excised omental gland proved it to be replaced by a highly cellular adenocarcinoma with no glandular arrangement whatever. Immediate recovery was uneventful, but the degree of glandular metastasis was so great that a radical operation was not performed, and the patient returned home.

During convalescence a test-meal was given and the free acid estimated at 3.5, the total acidity at 11.5 Ewald units. No organic acids were present. X-ray examination before discharge from hospital showed a six-hour residue of barium in the stomach, a large crater on the posterior wall, and some reflux of barium into the oesophagus.

Case 2—A gardener, aged 70, was admitted to hospital with a history of seven hours' severe, persistent, umbilical pain of sudden onset, accompanied by the vomiting of dark brown fluid. Epigastric pain, a few hours after food, and accompanied sometimes by vomiting, had troubled the patient at intervals for forty years, but had been recently improved by medical attention. The abdomen was markedly and diffusely tender and rigid. At immediate operation gas and gastric contents were present in the general peritoneal cavity, and a perforation through a large ulcerative prepyloric tumour was found. This was closed, and the peritoneal cavity drained. Recovery was satisfactory for sixteen days, after which the patient began to go downhill, and death occurred on the twenty-third day, probably from subphrenic abscess.

Case 3—A coal-miner, aged 45, was attacked suddenly a few hours before operation by a sharp stabbing epigastric pain with rigidity of the abdominal muscles. This sudden pain occurred while the patient was undergoing treatment in a medical ward for epigastric pain of two years' duration, increased by food, relieved by vomiting, and culminating in a recent hæmatemesis. The free acid had been estimated at 70, the total acidity at 100 in the fasting juice, 30 and 50 respectively, rising steadily for two hours, after test-meal. At operation a great deal of free fluid was found in the peritoneal cavity, and, in the stomach, a large saddle-shaped prepyloric ulcer, fixed and irremovable. The perforation was closed, an enlarged gland excised for examination from the lesser curvature, and the peritoneal cavity drained. The excised gland was shown microscopically to be replaced by masses of undifferentiated cells, with in scattered areas an attempt at gland formation. The patient made a good immediate recovery and left hospital on the eighteenth day of convalescence.

Case 4—A man, aged 65, was admitted with a subnormal temperature, an almost imperceptible pulse, and a cold clammy skin, too ill to give a clear history, but having suffered for twelve to eighteen hours from gradually increasing abdominal pain, mainly right-sided. Vomiting occurred once or twice. Operation was performed at once, pale yellow serous fluid found in the peritoneal cavity, and a large perforation, impossible of closure, discovered in a tumour of the lesser curvature. Jejunostomy was performed, but death occurred in forty-eight hours. The post-mortem examination revealed an early fibrinous peritonitis, from a perforated irregular ulcer of the lesser curvature. The stomach wall presented a hard diffuse infiltration around the ulcer, numerous secondary deposits were present in the omental glands and in the liver.

Case 5—A man, aged 57, was suddenly attacked by severe epigastric pain, unaccompanied by vomiting, and persisting without alleviation till admission to hospital six hours later. For fifteen years epigastric discomfort had been present, coming on half an hour after food, sometimes accompanied by vomiting of food, but relieved by sodium bicarbonate or the taking of more food. This history, supported by extreme rigidity of the upper abdominal muscles, encouraged a diagnosis of perforated gastric ulcer. At operation, which was performed immediately, gas and turbid fluid were evacuated from the peritoneal cavity. The parietal and visceral peritoneum was everywhere inflamed, and a perforation, $\frac{1}{2}$ in in diameter, was found on the anterior wall of the stomach, midway between cardia and pylorus, through an ulcerating tumour. Enlarged glands were present in the lesser omentum, but no other metastases were found. The perforation was closed, and gastro-enterostomy performed, but death followed within twenty-four hours.

Case 6—A man, aged 70, gave a history of severe lower abdominal pain of sudden onset and twenty-four hours' duration. The last meal taken had been vomited. After twelve hours there was some lessening of the pain, but its initial intensity was shortly renewed and surpassed. There had been one month's previous indigestion—slight epigastric pain one hour after food, unrelieved by alkalis or by the taking of more food. This had been accompanied by considerable loss in weight. Generalized abdominal rigidity was present, maximal in the epigastrium. Some tenderness was elicited in the hypogastrium and right iliac fossa, and the presence of free fluid in the peritoneal cavity was suspected. At operation yellowish fluid, suspending flakes of fibrin, was found in the peritoneal cavity. The whole stomach was generally infiltrated by malignant disease. The actual perforation was not with absolute certainty detected, but a leakage was suspected from a point on the lesser curvature. Enlarged glands were present both in the lesser omentum and in the gastrocolic ligament. Drainage of the peritoneal cavity was effected, but death occurred four days later. Section of an omental gland had all the appearances of a typical adenocarcinoma.

Case 7—A deaf-mute butcher, aged 72, was attacked suddenly by severe epigastric pain, vomited foul-smelling fluid once, and was admitted to hospital a few hours later. Difficulty of communication with the patient prevented any elicitation of an indigestive history, but he admitted some recent loss in weight. General tenderness and rigidity were present, maximal just to the right of the umbilicus. Slight distension was observed, no free fluid was apparently present, and no tumour was felt. On a diagnosis of perforated peptic ulcer, immediate operation was performed, turbid fluid was evacuated, and a perforation found and closed with difficulty. This had occurred through an obviously malignant ulcer of the anterior wall of the stomach which was partly adherent to the peritoneum of the abdominal wall. An enlarged gland was excised from the lesser curvature, but showed no malignant invasion. Death occurred forty-eight hours after operation, but a post-mortem examination was refused.

Case 8—A housewife, aged 56, gave a history of a severe attack, sudden in onset, of generalized, cramping abdominal pain, of eight hours' duration. For four years previously a heaviness had been complained of in the epigastrium, just to the left of the mid-line. No vomiting occurred. Food failed either to relieve or to increase the feeling of weight, which had no relation to meals. Recent loss of weight had been noticed. Epigastric rigidity and tenderness led to a diagnosis of perforated peptic ulcer, and immediate operation was performed. A large quantity of brown fluid found in the peritoneal cavity was evacuated, and a perforation through a carcinoma of the anterior wall of the pyloric antrum was closed with difficulty. No metastases were present. The patient made a good recovery and was discharged in three weeks, with the promise of a later gastrectomy, but on reporting later, a large mass already palpable in the epigastrium, and clinical evidence of liver metastases, forbade any further intervention.

ANALYSIS OF CASES OF PERFORATED CARCINOMA OF THE STOMACH

Chavannaz and Radoievitch,¹⁶ in a detailed and authoritative survey of perforated cancer of the stomach, with a collection from the literature of 46 cases, group the reported cases in four clinical categories: (1) Cases of sudden severe epigastric pain, accompanied by all the text-book signs of gastroduodenal perforation, and preceded by a history of indigestion; (2) Cases complaining of epigastric pain of similarly sudden onset, but with no previous dyspeptic phenomena; (3) Cases exhibiting the signs of a gradually progressive peritonitis; (4) Cases in which death occurs without attention being directed to the abdomen. It will be simpler, however, to merge the first two of Chavannaz's categories, which differ from each other only in the presence or absence of previous indigestion, and to combine also his third and fourth groups, since the fourth group may be considered merely as cases of gradual peritonitis insidious enough to escape the clinician's notice.

Of the 79 cases reviewed in the present paper, only 67 are reported complete with clinical histories. Of these, 44 (66 per cent) fall into the first group of fulminating cancerous perforation of the stomach into the general peritoneal cavity, and their histories correspond with those of seven of the cases reported above, and, incidentally, with the histories of typical perforated peptic ulcers. The remaining 23 (34 per cent of fully reported cases) share an absence of acute initial pain. In other respects their symptoms vary widely. These can be termed conveniently silent perforations of gastric carcinoma into the general peritoneal cavity, and among them is included the fourth case reported above. The 12 cases whose clinical histories are lacking are useless in an analysis of symptoms, but their operative and pathological findings will be referred to later.

I FULMINATING PERFORATIONS OF CARCINOMA OF THE STOMACH INTO THE GENERAL PERITONEAL CAVITY *

This, the commoner variety of malignant perforation of the stomach, includes two-thirds of the fully reported cases. The age incidence resembles that of carcinoma of the stomach, ranging from 23 to 78, 55 per cent of the cases fall in the 40-60 age period. Males suffer more than females in the proportion of 3 to 1.

All give symptoms now recognized as typical of gastroduodenal perforation—sudden severe epigastric pain often relieved spontaneously, but temporarily, after a few hours. The presence of vomiting is commoner than its absence, and hæmatemesis occurred in one case.⁶⁰ If operation is not performed, death occurs in one quarter of the cases within twenty-four hours of the onset of the pain, and one case in seven dies within six hours. Four cases lived longer than ten days without operation, and one lived for six weeks, in spite of an acute onset, a definite perforation, and, after death, the appearances of peritonitis. Of the 20 cases which came to operation, 70 per cent gave less than a twelve-hour history of pain. It is noteworthy that in half the cases perforation occurs while the patient is in bed, either at hospital or at home, under treatment usually for indigestion. In only one case is the patient specifically stated to have been up and about at the moment of perforation.

Previous Indigestion is extremely varied, but in long-standing cases a recent increase in severity is often observed. In only three cases is previous dyspepsia denied, in one-third of the cases it has been present for more than two years, and in one-sixth for more than five years. Previous hæmatemesis is more commonly admitted than denied.

Physical Examination elicits rigidity and tenderness suggestive of perforated peptic ulcer in four-fifths of the fulminant cases. A tumour mass is rarely palpated. The test-meal was examined in 8 cases, and in only 2 of these was achlorhydria present. In one case the acidity is described as normal, and in another free acid is stated to have been present, and total acidity to have measured 55 Ewald units. The remaining four cases gave figures of 3.5 and 11.5, 52 and 70, 74 and 92, 70 and 100.

The diagnosis of perforated carcinoma of the stomach was made in only 5 cases,^{27, 33, 40, 56, 62} and, of the remainder, fewer were diagnosed even as perforations

* Based on an analysis of 44 cases

than as such varied conditions as appendicitis, peritonitis, cholecystitis, internal hæmorrhage, or simple carcinoma with perforation unsuspected. In one case aortic aneurysm was the provisional diagnosis.

Operation was performed in 24 of these fulminating cases, of whom 11 left hospital alive—an immediate operative mortality of 54 per cent. If we exclude the cases of gastrectomy whose high recovery rate is probably artificial (*see below*), the immediate mortality rate rises to 68 per cent. This same high mortality rate seems to hold, no matter how early operation is performed.

Perforation is through the anterior wall of the stomach in nearly nine-tenths of the cases. In only two cases was perforation multiple. In one case³⁷ perforation occurred through an acute ulcer proximal to an annular tumour, and once¹⁷ perforation occurred through the anterior wall although the growth was on the posterior wall. The perforation is usually less than a $\frac{1}{2}$ in in diameter, but in one-sixth of the cases it is larger than a penny piece. In the enormous majority of cases the perforating tumour lies near the pylorus or on the lesser curvature, and in 84 per cent it is of the ulcerative variety. Metastases are present at the time of the perforation in nearly half the cases—usually in the omental glands, but sometimes in the liver, the pancreas, or the portal vein.

The pathological appearances of peritonitis are nearly always present shortly after perforation, and the exudate becomes seropurulent usually within twelve hours, occasionally the peritoneal cavity remains dry and free from severe congestion for some hours after perforation. The peritoneal exudate is not infrequently described as brown or black in colour.

The malignancy of the perforating tumour was confirmed histologically in approximately one-half of the cases, and in these the adenocarcinoma was the commonest histological variety.

II SILENT PERFORATION OF CANCER OF THE STOMACH INTO THE GENERAL PERITONEAL CAVITY *

This group includes one-third of all fully reported cases. The age and sex incidence corresponds to those of the fulminant variety. Only three of the silent cases had much pain, and even in these tenderness was slight or absent. The commonest course is an ascitic distension of the abdomen, with usually a dull generalized abdominal pain of gradual onset, and accompanied by little or no tenderness or rigidity. In one-fifth of the cases a palpable tumour is present. Occasionally^{42, 52} the only signs of perforation are sudden pallor, anxiety of expression, a rise in pulse-rate, a fall in temperature, and some slight abdominal tenderness. So far as can be ascertained, nearly two-thirds of the silent cases perforated while the patients were in bed in hospital undergoing treatment for gastric symptoms.

Previous Indigestion is of less than a year's duration in nearly nine-tenths of the cases in this group, and only three reported cases of silent perforation give a dyspeptic history of more than a year. Previous hæmatemesis is more often present than absent. No free acid was found in the two cases whose test-meal was examined.

* Based on an analysis of 23 cases

In only one of these cases was perforation even suspected, although in many a diagnosis of carcinoma of the stomach had been previously established. Tuberculous peritonitis, malignant peritonitis, and internal hæmorrhage are the most common misdiagnoses. Operation was undertaken in less than half of the cases, with an immediate post-operative mortality of 67 per cent.

The latent perforation, just as the fulminant, is usually solitary, and through the anterior wall of the stomach, although multiple perforations and posterior perforations occasionally occur.^{15 55, 66} The perforating tumour is almost invariably found in the pyloric region or on the lesser curvature. The ulcerative variety of cancer predominates, but proliferative and annular tumours are more common in this than in the fulminant series. Metastases in the gastric glands, the liver, or the lungs are already present in nearly half the cases at the time of perforation. Histologically, the adenocarcinoma predominates.

The peritoneal exudate is frequently purulent at the time of death or of operation, the peritoneum rarely shows marked congestion, and sometimes^{9 42} death supervenes before any peritoneal reaction occurs.

THE OPERATIVE TREATMENT OF PERFORATED CARCINOMA OF THE STOMACH

Theoretically, the treatment of choice in cases of perforated carcinoma of the stomach would perhaps appear to be the same as that adopted in perforated gastric ulcer, namely immediate closure of the perforation with or without drainage of the peritoneal cavity, and perhaps supplemented by gastro-enterostomy, to be followed by gastrectomy (in cases of carcinoma) at a later date. This treatment was in one instance adopted,²⁰ to leave the patient still alive and well ten years later. The presence of irremovable metastases in one half the cases of perforated carcinoma, however, abolishes in these any hope of a radical cure. Further, varying local conditions have necessitated in the reported cases a variety of technique at the initial operation.

The chief hindrance to simple closure of the perforation is the difficulty of apposing its friable malignant edges by sutures, and this sometimes suggests immediate gastrectomy as the only practicable procedure. The seven cases in which this line of treatment was adopted all made at least an immediate recovery, but it must be remembered that gastrectomy would be performed only in cases with the most favourable possible general condition, and there is a tendency probably for solitary gastrectomy successes to be reported, and for gastrectomy failures to be absent from the literature.

The recorded results of the various immediate operative procedures are best summarized in a table —

	Recovered	Died
Drainage alone	1	4
Closure alone	1	3
Closure and drainage	1	3
Local excision and closure	1	2
Closure and gastro-enterostomy	5	7
Closure and jejunostomy	—	2
Jejunostomy alone	—	1
Gastrectomy ^{10 28 41 61 62}	7	—
Total	16	22

PERFORATION IN 'ULCER-CANCER' AND IN PRIMARY CARCINOMA OF THE STOMACH *

It has been suggested that liability to perforation is a feature, and, indeed, an exclusive feature, of those cancers of the stomach which occur at the site of a previously existent simple gastric ulcer. This view is supported by the undisputed tendency of simple ulcer completely to destroy the muscular coat of the stomach, by the great preponderance (80 per cent) of ulcerating forms of carcinoma among perforated tumours, and by the perforating tumour's location, in 92 per cent of accurately reported cases, near the pylorus or at the lesser curvature. Of all perforated tumours, however, only 29 per cent complain of more than three years' previous indigestion, and 61 per cent have had dyspepsia of less than a year's duration. One case indeed,²⁸ in which a bismuth series six months before perforation failed to reveal any pathological lesion, establishes the occurrence of perforation in a recent cancer of a previously healthy stomach.

It is at this point convenient to make some effort to explain the occurrence of two separate varieties of malignant perforation of the stomach. We find that of the fulminant perforations only 46 per cent give a history of less than a year's indigestion, while 87 per cent of the silent perforations have had dyspepsia of this short duration. Further, of the eight fulminant cases whose gastric acidity was measured, while two had no free acid, one is stated to have had free acid present, one had free acid present in reduced amount, one had a normal free acid value, and three had definite hyperacidity. The two silent perforations whose test-meal had been examined both presented a low combined acid value, and an absence of free acid. This tendency of fulminating perforation to occur in cases with high gastric acidity and long history, of silent perforation to have a low acidity and a short history, at once suggests that the former may be a complication of ulcer-cancer, the latter a complication of primary carcinoma of the stomach.

The selection of cases of ulcer-cancer from a mass of published case reports is a difficult task—recognition of the condition is sufficiently puzzling and controversial when the whole clinical and pathological record of a case is directly accessible—yet in six of the reported cases (Friedenwald and McGlannan (2), Isch-Wall, Jaisson, Quenu, and Toms) a previous ulcer could be excluded by their short histories, their achlorhydria, and the lack of ulceration in the perforated tumour, perforation having in these cases presumably occurred from necrosis and sloughing of tumour tissue rather than from ulcerative destruction. Of these six cases, two³⁶ had a relatively painless course, and one⁵⁶ had a painful onset but little tenderness. The remaining three cases^{27, 34} had a painful course, but an early death (rapid even for a perforation) after two to five hours, from general peritonitis. The milder course in the first three of these is probably accounted for by the low acidity of the escaping gastric content, the rapidly fatal peritonitis in the second three cases by the admittedly septic content of many achlorhydric malignant stomachs.

* The term 'primary carcinoma of the stomach' is in the remainder of this paper used as a convenient title for those cancers of the stomach which are not preceded by simple peptic ulceration.

As a contrast to these, five cases (Albertoni, Thiede, Suter, Reiser, and the third new case reported above) could be justifiably labelled ulcer-cancer from their high gastric acidity, long histories, and strong resemblance to simple ulcer in appearance—malignancy in these was suspected only because of metastases, or because of their subsequent course (Three of them were definitely described as typical ulcer-cancers, but the difficulty of establishing a histological diagnosis of ulcer-cancer in a tumour whose muscle coat and muscularis mucosæ have been destroyed at the site of a perforation must be borne in mind) All these cases pursued a course identical with that of a perforated peptic ulcer

From these considerations it is perhaps justifiable to conclude that perforation of a primary carcinoma of the stomach is likely to be either relatively painless, mild, and gradual in its course, or, if sudden and painful, then rapidly fatal from septic peritonitis, it is certainly true that the symptoms of perforated cancer of the stomach will approximate closely to those of a typical perforated peptic ulcer, if the gastric acidity is high and the previous indigestion of long duration

GENERAL PERITONITIS IN CANCER OF THE STOMACH WITHOUT PERFORATION

Perry and Shaw, in a detailed consideration of perforated cancer of the stomach, describe 17 cases of carcinoma of the stomach that died of peritonitis, but in whom no actual perforation of the stomach could be found after death In 12 of the cases the onset of peritonitis was preceded by paracentesis or by an abdominal operation, but in the remaining 5 no such obvious route for infection was open By permission of Professor John Fraser I append notes of a similar fatal case of carcinoma of the stomach, with acute peritonitis, but without perforation —

A man, aged 46, complained of acute persistent boring epigastric pain, of sudden onset, and of twelve hours' duration Green sour fluid had been vomited, several years of 'indigestion' had preceded this attack The temperature was 100.8° and the pulse-rate 88, the whole abdomen was motionless and rigid, there was considerable general abdominal tenderness, but this was intense in the left upper abdomen No free fluid was suspected, and the liver dullness was not reduced On a diagnosis of perforated peptic ulcer laparotomy was performed, and the appearances of an acute peritonitis found A large tumour mass was present in the pyloric part of the stomach, but no perforation was present Fixity of the tumour and wide involvement of the omental glands prevented any attempt at gastrectomy, as also did the general condition of the patient A drain was accordingly passed down to the neighbourhood of the tumour, and the wound closed Death rapidly followed operation At autopsy the removed stomach was distended with water without any leakage occurring through the tumour or through the stomach above it The intestines were healthy and collapsed, the appendix was pale and free from disease, and no inflammatory focus was present elsewhere in the abdomen Histologically the tumour was of undifferentiated spheroidal cells

CONCLUSIONS

I Two-thirds of the cases of perforated carcinoma of the stomach are fulminant in their course, closely resemble perforated peptic ulcer in their symptoms and signs, and should be diagnosed as perforations of the stomach The only puzzling feature, present in one-fifth of the cases, is a relative or absolute absence of rigidity An exact diagnosis of perforated carcinoma is only likely if the gastric tumour has been previously diagnosed

2 In one-third of cases perforation is more or less silent, pain and abdominal rigidity being slight or absent Perforation should be suspected in cases of rapid ascites or collapse when a history suggestive of gastric carcinoma can be elicited

3 In both of these groups the perforating tumour is nearly always of the ulcerative variety, and situated on the lesser curvature or near the pylorus

4 In nearly half of all cases metastases are already present when perforation occurs

5 There is some evidence that a perforated ulcer-cancer of the stomach resembles in its symptoms and course a perforated peptic ulcer Primary cancer of the stomach tends to perforate atypically, being either mild and gradual, or else causing early death in a few hours from septic peritonitis

6 The immediate operative mortality rate closely approaches 60 per cent Immediate gastrectomy has been practised successfully in seven cases, excluding gastrectomy, the most effective form of treatment appears to be closure of the perforation, supplemented by gastro-enterostomy, and followed later by gastrectomy, if removal is practicable

7 Peritonitis can occur in the course of a case of cancer of the stomach without actual perforation of that organ

I am indebted to Mr J M Graham for permission to record the case which opens the present series, and which was treated in his wards The other original cases were collected by Mr L B Wevill and the Statistical Research Department of the Royal Infirmary, Edinburgh I wish to express my indebtedness also to Mr W J Stuart, Mr J W Struthers, Professor John Fraser, Dr Alexander Goodall, and Mr K Paterson Brown, for permission to use their cases

REFERENCES

- ¹ ACHARD, C, *Bull et Mem Soc med Hôp de Paris*, 1895, xii, 618
- ² ALBERTONI, P, *Arch ital Clin Med*, 1889 (cited by Essard)
- ³ AMBERGER, J, *Arch f klin Chir*, 1919, cx, 1106
- ⁴ AMBERGER, J, *Schweiz med Woch*, 1922 Feb 22 (cited by Suter)
- ⁵ ANDERS, J M, and MCFARLAND, J, *Proc Pathol Soc Philadelphia*, 1900, iii, 199
- ⁶ BALDWIN, A, *West Lond Med Jour*, 1902, vii, 28
- ⁷ BALLY, *Clin Hôp de Paris*, 1829, iv, 159
- ⁸ BARLOW and HABERSHON, *Med Times and Gaz*, 1862, ii, 325
- ⁹ BAUDET, *Bull et Mem Soc de Chir*, 1921, 212 (cited by Chavannaz)
- ¹⁰ BAUMANN, M, *Zentralb f klin Chir*, 1922, xlix, 1062
- ¹¹ BERNIER, A, *Contribution a l'Etude de la Co-existence de l'Ulcere et du Cancer de l'Estomac, et des Perforations de l'Estomac au Cours du Cancer*, 1902 Paris
- ¹² BRUNNER, *Munch med Woch*, 1922, lxix, 77
- ¹³ CADE and CROISSART, *Soc nat Med et des Sci med de Lyon* (cited by Chavannaz)
- ¹⁴ CASSAET, *Soc Anat et Phys* (Bordeaux), 1894, March 12 (cited by Chavannaz)
- ¹⁵ CHALIER, *Jour de Med de Lyon*, 1922, iii, 139
- ¹⁶ CHAVANNAZ, J, and RADOIEVITCH, S, *Rev de Chir* 1928, lxi, 111
- ¹⁷ CONTOUR, *Soc Anat de Paris*, 1840, xv (cited by Chavannaz)
- ¹⁸ CZERNY Cited by Chavannaz
- ¹⁹ DESTEFANO, J, and LIMA, E J, *Prensa med Argen* 1928, xv, 849
- ²⁰ DELAGINIERE, *Bull et Mem Soc de Chir*, 1923, xlix, 136
- ²¹ DIEULAFOY, *Presse med*, 1897, v, 289
- ²² VON EISELBERG, *Deut med Woch*, 1906, v, 2017
- ²³ ELLIS, *Boston Med and Surg Jour*, 1857, lvi, 481, 1860, lxii, 324
- ²⁴ ERTAUD, *Bull et Mem Soc de Chir*, 1914, xl, 224
- ²⁵ ESSARD, Thesis Lyons, 1906 (cited by Chavannaz)

- ²⁶ FALK, C C, *Int Jour Surg*, N Y, 1904, vii, 113
- ²⁷ FRIEDENWALD, J, and MCGLENNAN, A, *Amer Jour Med Sci*, 1919, cliii, 1
- ²⁸ GUIBAL, P, *Bull et Mém Soc nat de Chir*, 1930, li, 214
- ²⁹ HAMBURGER, W W, *Surg Clin Chicago*, 1917, i, 177
- ³⁰ HARDY, *Practicien*, 1881, iv, 614 Paris
- ³¹ HLMMETLR, J C, and AMLS, D, *Med Record*, 1897, lv, 362
- ³² HERARD DE BRESSI, *Gaz hebdomadaire de Med*, 1896, lvi, 1098
- ³³ HERZOG, E, *Zentralb f allg Pathol u pathol Anat*, 1928, lv, 437
- ³⁴ HORN, W, *Deut Zeits f Chir*, 1923, cli, 264
- ³⁵ HUTTON, *Proc Pathol Soc Dublin*, 1859, l, 167
- ³⁶ ISCH-WALL, *Bull Soc Anat*, 1889, lvi, 23
- ³⁷ JAISSON, *Arch de Mal de l'Appar digest*, 1913, lvi, 384
- ³⁸ KELLING, *Munch med Woch*, 1910, lvii, 1993
- ³⁹ KOSSANOVITCH, *Arch Serb de Med*, 1923, lvi, 49 (cited by Chavannaz)
- ⁴⁰ LAENNEC, *Rev méd franç et étrang*, 1824, i, 379
- ⁴¹ LAWEN, *Soc méd de Marburg*, 1921 (cited by Chavannaz)
- ⁴² LEJOINE and MILANOFF, Cited by Toinon
- ⁴³ LOPPER, M, and FORESTIER, J, *Bull et Mém Soc anat de Paris*, 1920, lv, 418
- ⁴⁴ LOYAL, Cited by Eiselsberg
- ⁴⁵ MAILLARD and BARBI, *Bull et Mém Soc anat de Paris*, 1913, lvi, 309
- ⁴⁶ MELLOT, *Rev méd franç et étrang*, 1831, iii, 34
- ⁴⁷ MESNARD, L, *Gaz hebdomadaire de Med de Bordeaux*, 1887, lvi, 566
- ⁴⁸ OZENNE, *Bull et Mém Soc anat de Paris*, 1880, li, 297
- ⁴⁹ PACKARD, *Trans Pathol Soc Philadelphia*, 1896, lvi, 55
- ⁵⁰ PAPIN, *Arch franco-belges de Chir*, 1926, lvi, 92
- ⁵¹ PEREIRA DE ROCHA, *Ann Soc anat-pathol de Bruxelles*, 1880, lvi, 161
- ⁵² PERRY, C, and SHAW, L E, *Guy's Hosp Rep*, 1904, lvi, 121
- ⁵³ PISSARY, COLOMBO, and SCHUTZENBERGER, *Bull Soc méd Hop de Paris*, 1913, lvi, 60
- ⁵⁴ PLANCIARD, *Soc anat Paris*, 1888 (cited by Jaisson)
- ⁵⁵ PRESSAT, *Bull et Mém Soc anat de Paris*, 1836, li, 60
- ⁵⁶ QUENU, Cited by Jaisson
- ⁵⁷ RAUZIER and ROGER, *Montpelier Med*, 1912, lvi, 514
- ⁵⁸ REITZENSTEIN, *Zeits f ärztlich Fortbild*, 1909 (cited by Chavannaz)
- ⁵⁹ ROLLET, *Lyon méd*, 1902, lvi, 526
- ⁶⁰ REISER, *Schweiz med Woch*, 1921, li, 923
- ⁶¹ SAVARIAUD, *Bull et Mém Soc de Chir*, 1922, lvi, 211
- ⁶² SUTER, *Schweiz med Woch*, 1923, lvi, 193
- ⁶³ TECQUENNE, *Bull et Mém Soc de Chir*, 1921, lvi, 349
- ⁶⁴ THIEDE, W, *Deut Zeits f Chir*, 1910, cli, 431
- ⁶⁵ TOINON, L, *Des Complications infectieuses des Cancres gastriques Perforans*, 1906 Lyon
- ⁶⁶ TOMS, S W S, *N Y Med Jour*, 1894, li, 141
- ⁶⁷ VAN DER CORPUT, *Ann Soc anat-pathol de Bruxelles*, 1869, lvi, 32
- ⁶⁸ WEST, C J, *Lancet*, 1888, i, 875
- ⁶⁹ WIDAL, Cited by Jaisson
- ⁷⁰ VON WINNIWARTER, *Wien klin Woch*, 1918, lvi, 620
- ⁷¹ ZUCCARELLI, *Marseilles med*, 1891, lvi, 815

BILATERAL BIPARTITE PATELLÆ

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A PATELLA in two parts is of scientific interest as an anatomical rarity and of practical importance in the differential diagnosis of fracture. This communication records an instance in which the condition was met with in both patellæ of a male dissecting-room subject aged 63 years. The fact that attention is not directed to the condition in most of the recent standard text-books of orthopædic surgery warrants this brief article.

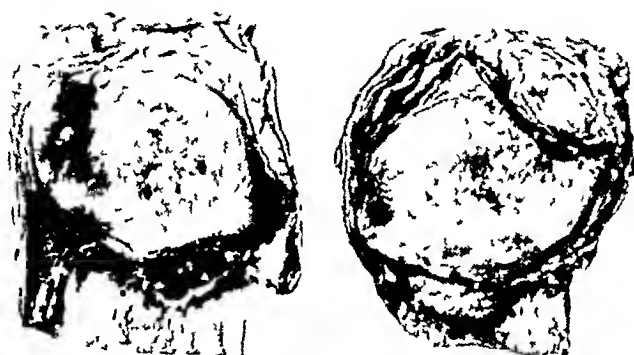


FIG 382—Posterior view of the patellæ. The supero-lateral portion of each patella is separate from the main portion. On the right side this separation has been made more evident by a knife-cut.



FIG 383—Radiogram of patellæ in Fig 382. The fragment at each supero-lateral angle is seen to be a separate piece of bone.

The gross appearances of the right and left patellæ are similar. At the supero-lateral angle of each patella a separate fragment is to be seen. It is oval in shape, about 2.5 by 2 cm in diameter, bony in structure, and separated from the main portion by a complete cleft (*Figs 382, 383*). The slight differences between the two fragments are that the right one is a trifle larger and more on the superior border, while the left one is a little smaller and more on the lateral border. Both of them, it will be noted, include the supero-lateral angle.

A bipartite patella can be, theoretically, the result of either an ununited fracture or the failure of two ossification centres to fuse with each other. It was thought that a microscopic examination of this specimen might assist in deciding which mechanism accounted for this particular example of the condition, so sections were

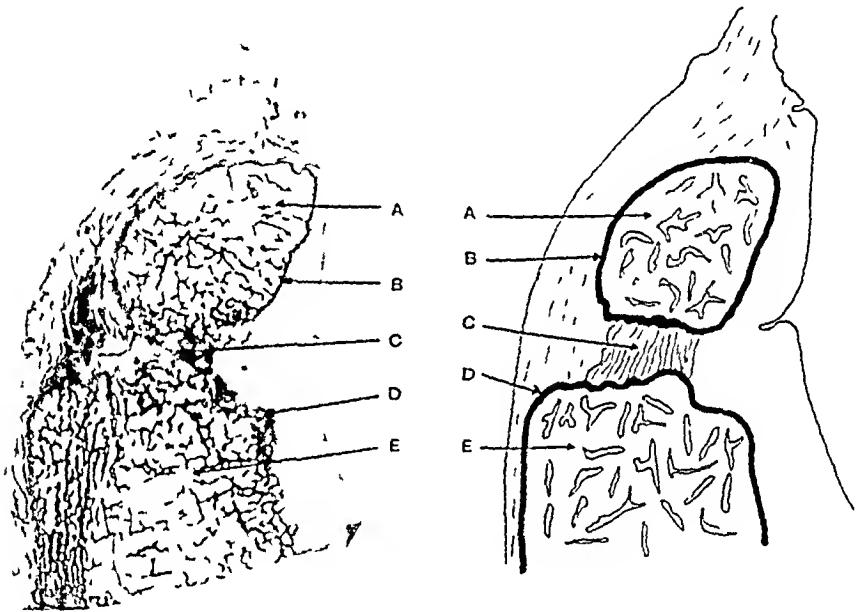


FIG 384—Low power photomicrograph and key sketch of section through the cleft in the bipartite patella. The separate portion is above, and the posterior or articular surface of the patella to the right. A, Cancellous bone of separate fragment of patella. B, Zone of old calcified cartilage. C, Fibrocartilage bond of union. D, Zone of old calcified cartilage. E, Cancellous bone of main portion of patella ($\times 2.6$).

cut through the two bony fragments and the tissue connecting them to each other. The sections were stained with hæmatoxylin and eosin, and certain points of interest were observed on examination (*Fig 384*).

The first point of interest concerned the condition of the articular cartilage, which covered not only each fragment but also the tissue in the area between them. The articular cartilage was continuous and showed no evidence of the repair that could be expected if there had been a fracture of the patella. As each bony fragment offered a foundation for the articular cartilage and was firmly joined to it, it seems almost inconceivable that a fracture of the bone could have occurred without a complete coincident fracture of the articular cartilage. A small fissure was present in the articular cartilage over the area joining the two bony fragments, but this fissure did not extend through more than half the thickness of the articular

cartilage There was no change in the deeper part of the articular cartilage in this area, and no substitution of connective tissue for cartilage in the area which would have represented the site of fracture in the cartilage if the patella had been fractured

The second observation of interest derived from an examination of the slides was the histological picture in the gap between the bony portions This gap was filled in with a type of connective tissue which demonstrated some of the features of both dense irregularly arranged fibrous connective tissue and cartilage The fibrous element was the predominant feature of the matrix, but occasional groups of cells, in lacunæ and surrounded by cell envelopes, indicated a cartilaginous heritage The bone was separated from this tissue by a line of demarcation similar to that surrounding the remainder of each bony portion Such lines of demarcation are familiar to those who study bone, and they arise because other tissues, such as cartilage, never become bone but are always replaced by bone Thus there is always a 'watermark' where bone formation encroaches on any other tissue

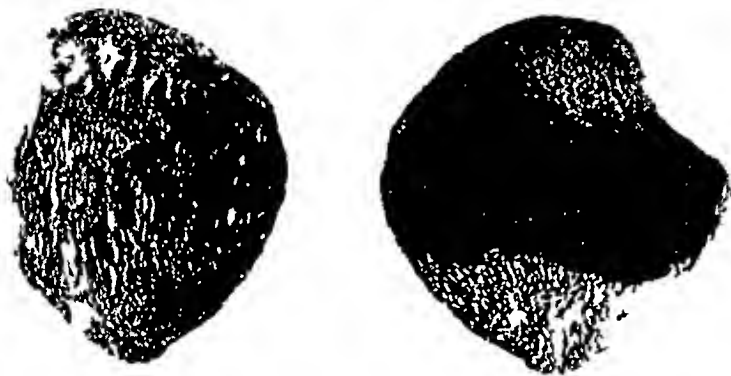


FIG 385—Though emargination of the patella appears not to have been described before 1902, its occurrence is no new thing, as this photograph of a specimen in Case 27 in the Natural History Museum at Torquay, England, exemplifies so excellently This patella was found with several other prehistoric human bones beneath the upper stalagmite deposit in Kent's Cave near Torquay It dates back probably to Magdalenian or Aurignacian times or earlier For this photograph I am indebted to Professor J C Boileau Grant and to Mr A H Ogilvie, F R A I, the Curator of the Museum

This watermark is often nothing more than a junction of two surfaces of different natures, a junction marked by the precipitation of stain This phenomenon often suggests the presence of a cement substance between the two surfaces, so that the watermarks are on occasion, perhaps none too accurately, called 'cementing lines' Lines of demarcation are intensified when bone replaces cartilage because the line is accentuated by the abrupt change in the staining reaction of the two types of matrix When the formation of bone from an ossification centre has spread throughout an area formerly cartilaginous, the spread of ossification is marked by an ever-widening circle or line of demarcation between the cartilage and the bone

Thus the picture in this area between the two bony portions of the patella suggests that the cause of the gap is the premature cessation of the spread of ossification from two separate centres The complete line of demarcation around each bony portion certainly suggests this explanation for the bipartite condition,

but cannot be considered as absolute evidence for this conclusion because it is possible that an abortive attempt at repair of a fracture might produce a line of demarcation between each bony fragment and the tissue which occupied the area between them. If, however, the latter phenomenon had occurred, there would be, it is thought, a histological picture of much irregularity in outline with other evidences of attempted repair, rather than a picture not dissimilar to that seen around the remainder of the periphery of each bony fragment.

In a search for additional specimens of bipartite patella, 153 dried patellæ from our bone collection were examined. One of these had a furrow on the articular surface about 5 mm from the lateral border. The area marked off by the furrow was penetrated on its anterior surface by a nutrient foramen, suggestive perhaps of an independent ossification centre. Seven other patellæ showed, in place of a supero-lateral angle, a depression first described by Kempson⁷ under the name of "emargination" (Fig 385). The other patellæ were found free from defects of the borders and showed no sign of a bipartite character.

A bipartite character is not peculiar to the patella, but is found in other bones such as the parietal, zygoma, medial cuneiform, and sesamoid of the great toe. Boyd² recently reported a pair of bipartite carpal navicular bones.

HISTORICAL REVIEW

In 1883 Wenzel Gruber,¹ of St Petersburg, described the first case of 'patella bipartita' and gave to the condition its name. He reported an autopsy on a man of 21 years in which he had demonstrated a patella, otherwise normal, with a small piece of bone attached by fibrous tissue to the supero-lateral angle. There the matter rested for nineteen years, until 1902, when Joachimstal² described a patella consisting of two bony parts, and Kempson⁷ noted that a depression at the supero-lateral angle was a common variation.

In 1904 Wright¹² described an anomalous patella in which two accessory patellæ included the greater part of the lateral border of the bone.

In 1919 Salmond⁹ described cases of fissured fracture of the patella in the neighbourhood of the insertion of the vastus lateralis. In the light of present knowledge there is little doubt that some of these cases were not fractures but anomalies of development.

In 1921 Wingate Todd and McCally¹¹ found some type of defect of the patellar border, such as fissure, fragmentation, or emargination, in 3 per cent of 682 skeletons. They explained all these conditions as phases of a single type of anomaly, that in which the patella is not ossified from a single centre but from two or more centres.

In 1925 George and Leonard³ reported 6 cases of persons having a separate piece of bone at the supero-lateral angle of the patella, and in 5 the condition was bilateral.

In 1930 Kahes⁶ reported the history and X-ray findings on 11 cases of bipartite patella. Ten had the separation at the supero-lateral angle and the eleventh was a very rare variety with a transverse cleft towards the distal part of the patella. The anomaly was unilateral in 4 of the 5 subjects in which X-rays were taken of both knees. In 1931 Neviaser⁸ reported a single instance of a unilateral bipartite patella.

EXPLANATION OF THE OCCURRENCE

Bipartite patella is generally stated to be an anomaly of ossification. Ossification of the patella begins in the second year and is complete about puberty. Ossification is usually from a single centre and rarely from two or more centres.

Adams and Leonard¹ and also Neviaser⁸ state that, even with two centres of ossification, the adult patella is usually one single bone unless fusion should fail to occur, in which case the result is bipartite patella.

Todd and McCally¹¹ suggested that an examination of the patellæ of children between the ages of 3 and 5 years might show more than one centre of ossification.



FIG 386 —X ray of the knee of a boy of 6½ years with two or three ossification centres for the patella, as indicated by an arrow

This was done by Shands¹⁰ in a study of the X-rays of the knees of 100 children between 2½ and 6 years of age. He found 2 children each with a patella having two definite centres of ossification. In one case the smaller centre was at the apex, in the other it was at the supero-lateral angle.

In following up this work of Shands, a study was made of the X-rays of 78 children's knees. None of these patellæ could be seen to be bipartite, but there is no record of the X-rays having been taken especially to give a clear patellar shadow, that is with the patella close against the film. One patella shows definite emargination at the supero-lateral angle. Another, from a boy of 6½ years, is of special interest (Fig 386). It appears to have three centres of ossification, though

there may be really only a single centre from which ossification is spreading irregularly. A second X-ray from the same child at 11 years shows no sign of bipartitism. This indicates that the presence of multiple centres of ossification in the young child is not always followed by the development of bipartite patella when development is complete. That is to say, multiple centres of ossification precede, but do not necessarily produce, a bipartite condition.

SUMMARY

In bipartite patella it is with rare exceptions the supero-lateral angle that is separate from the main portion of the bone. This condition seems to be due to the failure of an accessory centre of ossification to unite with the main portion of the bone. Bipartitism is mostly bilateral. Emargination is an allied condition, differing not in kind but only in degree.

In addition to its interest as an anatomical curiosity, there is the practical point that bipartite patella should not be mistaken for a fracture. The location of the crack at the supero-lateral angle will arouse the surgeon's suspicion, and the finding of a similar defect in the opposite patella will probably confirm it.

To Professor J. C. Boileau Grant and Dr. Arthur W. Ham the author is greatly indebted for helpful advice and assistance in the preparation of this report.

REFERENCES

- ¹ ADAMS, J. D., and LEONARD, R. D., *Surg. Gynecol. and Obst.*, 1925, *xl*, 601.
- ² BOYD, G. I., *Brit. Jour. Surg.*, 1933, *xx*, 455.
- ³ GEORGE, A. W., and LEONARD, R. D., *Amer. Jour. Roentgenol.*, 1925, *xxi*, 271.
- ⁴ GRUBER, W., *Arch. f. pathol. Anat.*, 1883, *xciv*, 358.
- ⁵ JOACHIMSTAL, *Arch. f. klin. Chir.*, 1902, *lxvii*, 342.
- ⁶ KALIES, W., *Diss. Tübingen*, 1930, 17.
- ⁷ KEMPSON, F. C., *Jour. Anat. and Physiol.*, 1902, *xxxvi*, 419.
- ⁸ NEVIASER, J. S., *Ann. of Surg.*, 1931, *xciv*, 150.
- ⁹ SALMOND, R. W. A., *Brit. Jour. Surg.*, 1919, *vi*, 463.
- ¹⁰ SHANDS, A. R., *Jour. Bone and Joint Surg.*, 1926, *viii*, 824.
- ¹¹ TODD, T., WINGATE, and MCCALLY, W. C., *Ann. of Surg.*, 1921, *lxxiv*, 775.
- ¹² WRIGHT, W., *Jour. Anat. and Physiol.*, 1904, *xxxviii*, 65.

EXPERIMENTAL SURGERY

OSTEITIS FIBROSA: AN EXPERIMENTAL STUDY

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INTRODUCTION · BIOCHEMICAL CONSIDERATIONS

UNTIL recent years the group of diseases classified clinically as the osseous dystrophies has remained outside the field of exact pathology. The group includes von Recklinghausen's osteitis fibrosa with cysts and giant-celled tumours, localized osteitis fibrosa which may be single or multiple, Paget's osteitis deformans, rickets, renal rickets, 'delayed' rickets, coeliac rickets, osteomalacia, and various idiopathic forms of osteoporosis. Although it is now generally recognized that each of these conditions has its own specific pathology, yet it must be remembered that they also have the common character of the decalcification of the skeleton. It is therefore by the study of the calcium metabolism in these different disorders that the mechanism of ossification and decalcification, both physiological and pathological, is being elucidated. Therefore, although the main object of this paper is to investigate the process by which hyperparathyroidism produces the bony lesions which have come to be recognized as characteristic of its action, it is necessary first to consider osteitis fibrosa in relation to the general metabolism of calcium.

The skeleton, besides being the framework of the body, is the reservoir of calcium for the organism, which is constantly being drained or built up, according to the supply of available calcium in the serum, and the mechanical requirements of the bones themselves.

Calcium differs from most other important metabolites, such as sugar or chloride ions, in that there is no conservation of it in the body, and deprivation of calcium results in a constant drain of it from the skeleton into the urine and faeces, in excess of the amount absorbed—a so-called negative calcium balance.

Lack of calcium may be due to an actual insufficiency in the diet, or to deficient absorption from the intestine in spite of an adequate diet, or may be the result of excessive excretion of calcium salts. Calcium can only be absorbed from the intestine in soluble form¹. Excess of phosphate or alkali (carbonate) in the diet precipitates the calcium in the gut and prevents its absorption, while incomplete digestion and absorption of fats causes precipitation of calcium soaps in the intestine with the same result. Similarly, lack of vitamin D, which is necessary to the absorption of fats, causes insufficient absorption of calcium, and, as calcium salts tend to be adsorbed on to protein molecules, it may well be that incomplete protein absorption from the gut, by minimizing calcium absorption, is a factor in the etiology of 'idiopathic' forms of osteoporosis.

As in absorption, so in excretion, calcium is influenced by variations in the metabolism of other substances, the effects of which are reflected in the calcium content of the serum. The serum calcium must be regarded as a solution in 'biological saturation' with the calcium phosphate of the bones and is normally 9 to 11 mgrm per 100 c c serum. This biological saturation is greatly in excess of the ordinary chemical solubility of calcium phosphate and is dependent on three factors—the concentrations of protein and of phosphate in the serum, and the amount of parathyroid hormone present.

Part of this excess calcium in the serum is due to adsorption of calcium salts on to the serum proteins. The amount adsorbed varies directly with the amount of protein present, 1 mgrm of protein being equivalent to approximately 0.5 mgrm of calcium per 100 c c serum, so that normally 2.5 to 4.5 mgrm of the total serum calcium can be ascribed to this factor.² The adsorbed calcium, however, is not ionized and is physiologically inactive—for example, the reduction of the serum proteins which occurs in certain forms of nephritis, with consequent reduction of the adsorbed calcium, does not cause tetanic symptoms.

The remainder of the serum calcium is present as calcium ions formed by dissociation of the calcium phosphate of the bones, but the amount, 6 to 7 mgrm per 100 c c, is still in excess of the normal solubility of calcium phosphate, 2.5 mgrm per 100 c c. This excess in ionized calcium is due to the influence of the parathyroid hormone.³ The mechanism of this action is not fully understood, but not only has it been shown that the ionized calcium of the serum is reduced after parathyroidectomy, and is restored by the administration of parathormone, but that hypercalcaemia can be produced by excess of parathormone, and that the degree of hypercalcaemia is proportional to the amount of parathormone given.

Although the dissociation of the calcium phosphate of the bones into calcium and phosphate ions of the serum is promoted in this way by the parathyroid hormone, the dissociated ions and the solid calcium phosphate of the bones are still in equilibrium. They are therefore subject to the law of ionic dissociation—namely, that the concentrations of the calcium and phosphate ions, if altered, must vary inversely with each other, in order that they may remain in equilibrium with the undissociated calcium phosphate.⁴ Consequently, excess of phosphate ions in the serum from other sources causes a fall in the serum calcium, with deposition of calcium phosphate in the bones, while a reduction of phosphate in the serum is accompanied by a rise in serum calcium, and solution of calcium phosphate from the bones.

Only this ionized part of the calcium in the serum is physiologically active, and reduction of the ionized calcium by parathyroidectomy or by excess of phosphate ions will cause the symptoms of tetany to appear. (In forms of nephritis other than those referred to above, where phosphate is retained in the serum, the concentration of calcium ions may be reduced to such an extent as to cause tetany.)

It must be noted that although the protein-adsorbed calcium is inactive, it is necessary to determine the proportion of the total serum calcium thus put out of action in any particular case, before it is possible to estimate the amount of active calcium present. In the investigation of a case of osteoporosis, therefore, the significance of an abnormal serum calcium can only be interpreted by a knowledge of the serum proteins and phosphate.

There are thus two main factors which control the balance of calcium between the serum and the bones, the concentrations of phosphate ions and of parathormone.

in the circulation. Turning first to the influence and variations of the phosphate, we find ourselves dealing at once with the acid-base metabolism of the body, because the phosphate radical is one of the most important 'buffer ions' in the tissues. If acidosis is impending, the buffer phosphate is excreted and the serum phosphate falls, calcium phosphate is then dissolved from the bones to restore ionic equilibrium in the serum, and the consequent excess of calcium in the blood is excreted by the kidney. Thus acidosis, which occurs in so many conditions, may result directly in a drain of calcium from the skeleton into the urine. (A distinction must be made between the acids present in the gut which are necessary to the normal absorption of calcium, and the acidosis produced by faulty metabolism, which may cause decalcification of the skeleton.)

Calcium metabolism, then, is dependent on many outside conditions and circumstances—acidosis with all its causes, the metabolism of fat and protein, vitamin D effects, and errors of dietary. All these finally affect the calcium reserves of the body, and to them the decalcification of the skeleton observed in many clinical cases, can be directly attributed. In rickets and coeliac rickets the skeletal effect is due entirely to lack of calcium in the serum, in renal rickets it is due to phosphate retention by the kidney, with consequent excretion of calcium phosphate by the gut (see Case 1, p. 585), while osteomalacia is the combined result of diminished absorption of calcium following vitamin lack, and the acidosis of malnutrition and a cereal diet, together with the extra drain on the calcium reserves by the foetus in utero.

All cases of decalcification cannot, however, be explained by these phenomena alone. Two advances have been made in recent years which have gone far to the elucidation of the problem—the discovery of phosphatase by Robison in 1924,⁵ and the preparation of an active extract of the parathyroid glands by Collip in 1925.⁶ The discovery of parathormone has placed in our hands not only the explanation of the apparent supersaturation of calcium in the serum, but also has helped us to recognize that now familiar type of von Recklinghausen's disease consequent on a parathyroid adenoma. It is to be noted, however, that decalcification by parathormone proceeds by the same mechanism as by acidosis. Thus Albright, Bauer, and their colleagues⁷ have shown that the first action of parathormone is to sweep phosphate from the blood into the urine, the serum phosphate falls, consequently the serum calcium rises, and reserves of calcium and phosphate are mobilized from the bones. The excess of calcium is excreted by the kidney, more is supplied from the bones, and so a drain of calcium from the skeleton into the urine is established. Decalcification, therefore, whether by parathormone or acidosis, is the result of excretion of phosphate. The difference, however, between the two agents is that in the case of parathormone there is also an *absolute* hypercalcaemia, due to the increase in solubility of the calcium phosphate in the bones, while in acidosis the calcium in the serum is only increased relatively to the phosphate.

Fatal doses of parathormone result in an increase of phosphate as well as calcium in the serum shortly before death, so that parathormone may have a direct action on the calcium of the bones in these circumstances. This action, however, does not come into play with other than lethal doses. Toxic doses cause weakness, hypotonia of the muscles, and anorexia, followed by vomiting, coma, and death. The apparent toxicity, however, varies with the amount of calcium in the diet, and much larger doses of parathormone can be tolerated by an animal on a low calcium diet, where the hypercalcaemia is not so extreme, than by an animal on a high calcium

diet.⁸ The toxic element is, therefore, the hypercalcaemia rather than the parathormone—a point of some importance in connection with the experimental results in this paper.

In the human subject there can be no doubt that the hyperparathyroidism consequent on a parathyroid adenoma does cause von Recklinghausen's disease, and it has been repeatedly shown that removal of the adenoma causes the bones to return gradually to normal. However, not all parathyroid tumours have von Recklinghausen's disease—40 per cent of the cases of parathyroid adenomata collected by Barr and Bulger⁹ had no bony lesion. Further, many cases of generalized osteitis fibrosa established by histological examination of a piece of bone removed for section have shown, on biochemical investigation, no evidence of hyperparathyroidism. In not a few such cases a thorough exploration of the neck has failed to demonstrate a tumour, and even normal parathyroids have been removed without effect on the bones. Also we have seen that the skeleton is liable to decalcification by such conditions as acidosis, in the same way as by parathormone, at any rate in ordinary doses. Considering all these facts, it is essential at the present stage of our knowledge not to regard the histological features of von Recklinghausen's disease and hyperparathyroidism as specifically related. The characteristic features of von Recklinghausen's disease are, clinically, that it is seen in fairly young adults, although the process actually began at some time in youth when the bones were most plastic, while, histologically, the presence of the cysts and the giant-celled tumours distinguish it from other decalcifying diseases. The question is: Are these histological characters necessarily the result of the action of parathormone, or can it produce decalcification without them? Conversely, can giant-celled tumours and cysts appear with decalcification from some other cause than hyperparathyroidism? Can von Recklinghausen's disease, complete with giant-celled tumours and cysts, be produced experimentally by the action of parathormone alone? The second part of the experimental work of this investigation is concerned with this problem.

The other discovery, that of phosphatase, has helped us to understand the chemical processes of ossification. Phosphatase is an enzyme that hydrolyses organic phosphates into inorganic phosphates, and it occurs at the three principal sites of phosphate metabolism—namely, in the intestinal mucosa where phosphorus is absorbed, in the bones where it is stored, and in the kidneys where it is excreted. The enzyme, however, is only present in these tissues when and where they are actively functioning. There is practically no phosphatase in the foetal kidney, and the phosphatase of bone occurs only at the sites of active ossification,¹⁰ at the epiphyseal line and under the periosteum. It appears, in fact, to be produced by, or concentrated in, the osteoblasts in these situations. It is this local concentration of phosphatase in the osteoblasts that determines ossification, ionized inorganic phosphate is produced *in situ* from the practically non-ionized organic phosphates of the serum. This local increased concentration of phosphate ions upsets the local calcium-phosphate equilibrium, so that the solid salt is precipitated around the osteoblast. In old compact bone with widely separated bone-cells the calcium is precipitated in lamellae round the osteoblasts, but in an area of newly forming bone with young osteoblasts crowded together, the calcium is precipitated as a network of fine fibrils in the tissue matrix. This physiological process of calcification to form bone is quite different from the process of pathological calcification,

such as occurs in tuberculous glands or arterial walls. The latter type of calcification is always preceded by *degeneration* of the tissue cells as the result of local toxins and/or local oxygen deficiency. The lipid substances liberated in the process of degeneration form compounds analogous to calcium soaps, which gradually change into deposits of phosphate and carbonate of calcium.

This is the explanation of the metastatic calcification observed in experimental hyperparathyroidism, where toxic doses of parathormone have been given.¹¹ This metastatic calcification has clouded the conception of calcium mobilization by parathormone, for apparently excess of parathormone in the circulation causes actual deposition of calcium in the soft tissues. The toxic effects of hypercalcaemia mentioned above have, however, been observed by many workers, and McJunkin¹² has shown that the metastatic calcification in the soft tissues is always preceded by marked degenerative changes. This effect, then, is an example of pathological calcification, the calcium being precipitated from the serum, primarily in combination with lipid bodies, independently of the presence or absence of parathormone.

It is the phosphatase in the osteoblast, then, that brings about ossification. Now it has been discovered that in von Recklinghausen's disease and Paget's disease and other disorders with extensive decalcification of the bones, there is a marked increase in the concentration of phosphatase in the plasma.¹³ Normally the concentration in the plasma is very low, 0.06 units per cubic centimetre, but in these generalized diseases of bone the concentration of phosphatase in the plasma may be increased as much as twenty times or more. The questions arise: What is the explanation of this raised plasma phosphatase? Is it connected with the cause or the result of the disease process in the bones? What is the function and fate of the phosphatase in this high concentration in the blood? Is it destroyed or excreted? The first part of the experimental work set out below deals with these problems.

EXPERIMENTAL WORK

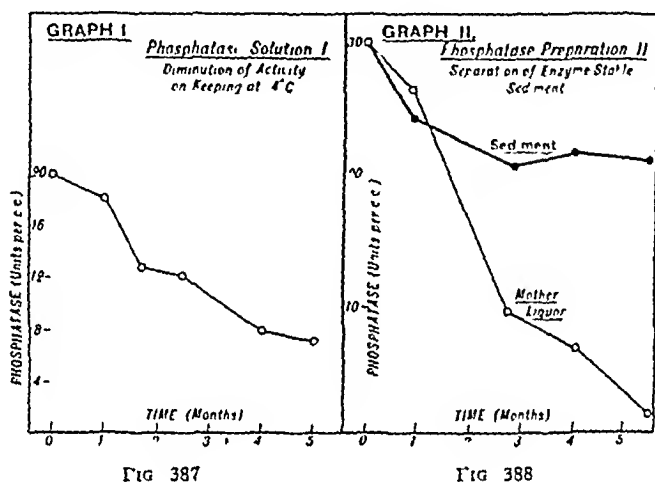
PART I—EXPERIMENTS WITH PHOSPHATASE

Preparation—A sterile solution of phosphatase, suitable for injection into rabbits, was made by a modification of Kay's method as follows. Two warm fresh kidneys enclosed in the perinephric fat, to avoid bacterial contamination, were obtained from a recently killed pig, the organs were dissected out from the fat with sterile instruments, and the renal capsules removed. The sterile kidneys were then dissected so that the cortex was roughly separated from the medulla (the phosphatase occurs almost exclusively in the cortex of the kidney, which is also much more easily crushed than the whole kidney with its fibrous calices). The cortex was then thoroughly ground up with sterile sand and chloroform water in a sterile mortar, and allowed to stand for twenty-four hours with repeated stirrings at room temperature. At the end of this time the fluid extract, containing about 90 per cent of the total phosphatase of the kidney substance, was collected and kept in an ice-chest, and subsequently was used for injection into the rabbits.

Estimation. Diminution of Activity on Keeping—Kay's method and unit were also adopted—namely, a known quantity of solution containing the

unknown amount of enzyme was allowed to act on a substrate of excess of sodium-B-glycero-phosphate at a P_{H} of 7.6 for forty-eight hours at 38°C . The concentration of phosphatase was then calculated from the amount of inorganic phosphate liberated from the organic combination by the action of the enzyme, and the unit was expressed as that amount of phosphatase which, acting on excess of substrate under the above conditions, liberated 1 mgrm of inorganic phosphate in forty-eight hours.

In all, three solutions of phosphatase were prepared during the course of the work, and it became necessary to estimate the strength of the solutions from time to time in order to determine dosage. The strength of the first solution was 20 units per cubic centimetre when first made, and Fig 387 shows the rate at which the activity diminished over a period of seven months at -4°C .



The second preparation of the enzyme was made some time later. It was richer in phosphatase (30 units per cubic centimetre) and it was found that on standing for a long time at -4°C a whitish sediment very slowly separated out. On investigation it was discovered that while the phosphatase activity of the mother liquor gradually diminished, that of the sediment remained constant. The precipitate was, in fact, a stable preparation of phosphatase. Fig 388 illustrates the relative activities of liquor and sediment with the lapse of time.

Further investigation was instituted on this property of the kidney extract. It was found that the sediment could not be extracted from the fresh preparation by ordinary centrifuging or by filtering under pressure. However, on centrifuging at 30,000 revolutions per minute in a Sharple's centrifuge, a greyish-white substance gradually adhered to the sides of the vessel. This glutinous material was scraped off with a little distilled water and the resulting emulsion was found to be an extremely concentrated preparation of phosphatase, as much as 36 units per cubic centimetre. The chemical nature of the sediment was not exactly determined, but it gave the qualitative tests for protein. The enzyme was probably adsorbed on to the protein debris of the crushed renal cells.

Injection of Phosphatase into Rabbits—The standard enzyme solution prepared, it was injected into rabbits to determine whether the plasma phosphatase could be raised by this means, and, if so, whether any lesion in the bones would

follow The object of this procedure was to determine whether the high plasma phosphatase observed in decalcifying diseases is concerned with the causation of the condition, or whether it is a secondary result of the disease process

Experiment 1—Two adult rabbits on an adequate calcium diet were X-rayed, with controls, and their blood calcium, phosphorus, and phosphatase were determined They were then given daily injections of the first solution of phosphatase, in increasing doses up to 65 units intramuscularly Neither animal died as a result of this, and estimations of the serum calcium, phosphorus, and phosphatase were made at intervals for six weeks At the end of that time, little effect having been noticed either in the blood chemistry or X-ray appearances, the method of administration was changed to the intravenous route, whereupon one animal died, and the other was injected with smaller doses for eight days more, and was then killed

Table I shows the dosage of phosphatase, and the values of the serum calcium and the plasma phosphorus and phosphatase, the two sets of figures referring to the different rabbits

Table I

DATE AND CALCULATED UNITS OF PHOSPHATASE INJECTED	SERUM CALCIUM	SERUM PHOSPHORUS	PLASMA PHOSPHATASE
	Mgrm per 100 c c	Mgrm per 100 c c	Units per c c
April 19	R ₁ 14.4 R ₂ 14.8	4.4 4.9	0.16 0.16
Increasing daily dose up to 65 units intramuscularly			
April 30	R ₁ 15.0 R ₂ 14.2	5.2 5.0	0.16 0.14
Daily dose of 6 c c extract—equivalent to 62 units, falling to 47 units of phosphatase			
May 31	R ₁ 13.8 R ₂ 15.2	5.4 4.8	0.14 0.19
Rabbits X-rayed Intravenous dose up to 15 units daily			
June 8	R ₁ 13.6 R ₂ died	3.8	0.14

It will be seen that the injections caused no effect on the serum calcium or phosphorus outside the normal variations for the rabbit, nor was the phosphatase itself raised in the plasma Post-mortem examination showed some suppuration at the site of injection, but no softening of the bones or calcification of the viscera, which were normal in all respects Sections were cut of the ribs, femur, and tibia, but showed no alteration from the normal bone histology

Apart from the lack of bone change, it seemed extraordinary that the level of phosphatase in the plasma should remain normal, in spite of continued injection of the enzyme Therefore it was decided to inject much larger doses in further rabbits, using the second stronger preparation of phosphatase, to see whether or not it was possible to raise the plasma phosphatase in this way

Experiment 2—After preliminary chemical investigation of two rabbits as before, 10 c c of emulsion of the phosphatase sediment, equivalent to 200 units,

were given intramuscularly daily for five days. Estimations were made of the blood calcium, phosphorus, and phosphatase on the second and fifth days, the blood being taken five hours after the last injection. Again (*Table II*) only very slight alterations in values were found, and as before no pathological effect was discovered in the soft tissues or decalcification in the bones.

Table II

	SERUM CALCIUM	SERUM PHOSPHORUS	PLASMA PHOSPHATASE
	Mgmm per 100 c c	Mgmm per 100 c c	Units per c c
Before injection	R ₁ 16.0 R ₂ 15.5	4.4 5.0	0.12 0.14
200 units of phosphatase injected daily			
2nd day	R ₁ 15.8 R ₂ 15.5	4.8 5.4	0.15 0.16
5th day	R ₁ 16.2 R ₂ 15.8	4.8 5.0	0.14 0.14

The negative results of these two experiments raised the question whether phosphatase prepared from a pig's kidney was active in a rabbit, and, if so, what was the reason for the rapid disappearance of such large amounts so soon after injection. The third experiment was therefore made to discover the fate of the phosphatase injected, whether excreted in the urine, or destroyed in the circulation. It was first necessary to estimate the phosphatase content of the urine. In the human this is a very difficult matter, because the large amount of inorganic phosphate present renders the extra amount produced by the phosphatase insignificant in comparison, the difference being well within the limits of experimental error. M. C. Bourne,¹⁴ however, has reported that rabbits on a diet of cabbage only, excrete practically no phosphate in the urine. This point was investigated, and it was found that rabbits fed on a daily diet of 150 gm of cabbage and 75 gm of oats excreted only a minute amount of phosphorus in the urine. Consequently, it was possible to apply the ordinary technique to the estimation of phosphatase in the rabbits' urine.

Next it was necessary to determine whether phosphatase in the urine was stable, or whether its activity was in any way inhibited. To decide this point, the following experiment was performed —

A series of five flasks was prepared containing (1) Normal rabbit's urine, (2) Concentrated phosphatase solution, (3) Diluted phosphatase solution (1:10), (4) A mixture of 1 and 2, (5) A mixture of 1 and 3. Phosphatase estimations were performed on these five solutions, at intervals up to twenty-four hours later, and the results are set out in *Table III*.

From *Table III* the following facts can be deduced (1) That phosphatase is normally excreted in small amounts in the urine (column 1). (2) That on standing this phosphatase is fairly stable in the urine (column 1). (3) That although the activity of the concentrated solution of phosphatase in urine, as in water, does decrease slightly on standing, the dilute solution is relatively stable (column 5). As the phosphatase in the urine of the rabbits in the main experiment which follows

is dilute, the results obtained may be regarded as accurate (4) That the activity of phosphatase is not inhibited by the urine, as the phosphatase values in column 4 are roughly the means of columns 1 and 2, and those in column 5 are the means of

Table III

HOURS AFTER MIXING URINE AND PHOSPHATASE	PHOSPHATASE VALUES (UNITS PER C C)				
	(1) Rabbits' Urine	(2) Phosphatase Solution Conc	(3) Phosphatase Solution Dilute	(4) Mixture of 1 and 2	(5) Mixture of 1 and 3
0	0 20	0 74	0 19	0 57 0 47	0 27 0 20
1½	0 19	0 71	0 18	0 55 0 48	0 24 0 19
7	0 22	0 55	0 19	0 51 0 39	0 27 0 21
24	0 18	0 61	—	0 46 0 40	—

The italic figures in columns 4 and 5 are the calculated arithmetic means of the phosphatase values of columns 1 and 2 and 1 and 3 respectively, for comparison with the phosphatase values in the mixtures of urine and phosphatase solutions actually found

columns 1 and 3 The constant slight excess of the figures in columns 4 and 5 over the calculated amounts is due to the relatively increased activity of the enzyme by the dilution of the phosphatase solution with the urine (compare columns 2 and 3) This property is common to all enzymes

Experiment 3 Fate of Injected Phosphatase in the Body—An adult rabbit, fed on the same diet as in the preceding experiment, was put in a metabolism cage

Table IV

TIME	PLASMA PHOSPHATASE	PLASMA PHOSPHORUS	URINE PHOSPHATASE	URINE PHOSPHORUS	VOLUME URINE IN PERIOD
	Units per c c	Mgmm per 100 c c	Units per c c	Mgmm per c c	c c
9 50 a m	0 047	3 2	—	—	—
10 50 a m	—	—	0 263	0 55	3
11 0 a m	Injection of 580 units of phosphatase				
11 35 a m	0 061	—	—	—	—
12 35 p m	—	—	0 342	0 52	4
1 0 p m	0 070	4 6	—	—	—
1 30 p m	—	—	0 098	0 26	23
2 15 p m	—	—	0 160	0 48	6 5
2 35 p m	0 054	—	—	—	—
5 0 p m	0 039	4 0	0 132	0 56	11 5
9 40 p m	0 048	2 8	0 142	0 56	24
9 40 a m	0 055	2 8	0 095	0 46	64

After emptying the bladder, phosphorus and phosphatase estimations were made on the urine, and on a sample of blood taken from the ear A massive single dose of 580 units of phosphatase (Sharple's centrifuge preparation) was then injected

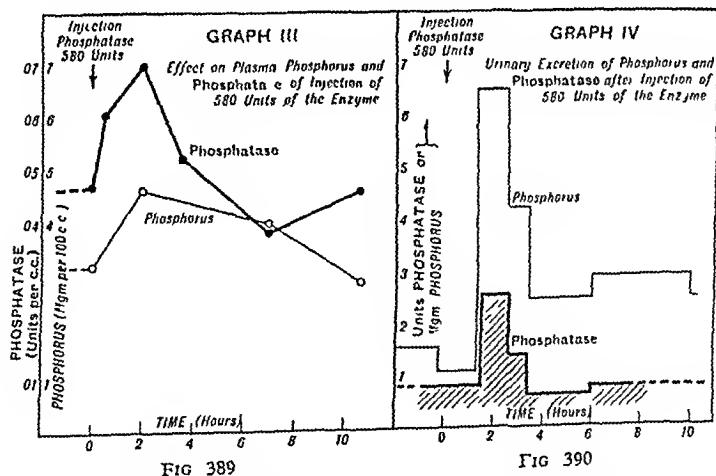
intramuscularly at four different places. From an hour to an hour and a half later, the animal looked ill and had occasional tremors, but subsequently recovered. Specimens of blood were taken at intervals for the next twenty-four hours, and the urine collected at the same times. Estimations of phosphorus and phosphatase were made on both blood and urine, and the volume of urine in each period was measured. The results are shown in *Table IV*, and from these data the actual amounts of phosphorus and phosphatase excreted in the urine in each period were calculated and set out in *Table V* (columns 2 and 3). From these figures and the duration of each

Table V

PERIOD	TOTAL PHOSPHATASE EXCRETED DURING PERIOD	TOTAL PHOSPHORUS EXCRETED DURING PERIOD	RATE OF EXCRETION OF PHOSPHATASE	RATE OF EXCRETION OF PHOSPHORUS
9 50-10 30 a.m.	0.79	1.65	Units per hour 0.79	Mgms. per hour 1.65
Injection of 580 units of phosphatase				
10 50-12 35 p.m.	1.37	2.08	0.79	1.2
12 35-1 30 p.m.	2.25	6.0	2.45	6.5
1 30-2 15 p.m.	1.04	3.12	1.39	4.2
2 15-5 0 p.m.	1.52	6.49	0.55	2.4
5 0-9 40 p.m.	3.41	13.52	0.73	2.9
9 40 p.m.- 9 40 a.m.	6.10	29.20	0.51	2.4

Twenty-four-hour total of phosphatase excreted = 16.4 units (as against 580 units injected)
(— Summation of Column 2)

period, the rates of excretion of the two substances were calculated (columns 4 and 5, *Table V*). *Figs 389, 390* were constructed from the data in *Tables IV and V* and show respectively the plasma values and the rates of excretion of phosphatase and phosphate during the twenty-four hours after the injection. From *Fig 389*



it can be seen that the plasma phosphatase is at a maximum two hours after the injection, presumably the height of absorption into the circulation, and that it falls to normal again three hours later. The corresponding rise in plasma phosphorus and the coincident increased excretion of phosphatase (*Fig 390*) indicate that while

the phosphatase is in the tissues it is actively producing inorganic phosphate from organic combination. In other words, phosphatase prepared from a pig's kidney is active in a rabbit.

The tremors (? incipient tetany) observed in the rabbit an hour and a half after the injection also correspond to the rise in plasma phosphorus at this time, and may possibly be due to a lowered serum calcium attendant on this. Unfortunately calcium estimations were not done in this experiment.

The rapid return of the plasma phosphatase to normal after its temporary rise explains the inability to find a raised plasma phosphatase in the first two experiments, as blood samples were taken five hours or more after the injections of the enzyme.

Fig. 390 shows a delay of one and a half hours in the excretion of phosphatase, after its rise in the plasma, followed by a period of two hours of increased excretion, after which excretion also returned to the normal rate. A consideration of *Table V*, however, shows that the actual total amount of phosphatase excreted in the twenty-four hours after the injection was only 16.4 units, whereas 580 units were injected. By far the greater proportion of the injected phosphatase must therefore have been destroyed in the body.

SUMMARY OF RESULTS OF PHOSPHATASE EXPERIMENTS

- 1 Phosphatase is normally excreted into the urine in small amounts.
- 2 Phosphatase prepared from a pig's kidney is active when injected into a rabbit.
- 3 Phosphatase injected into a rabbit over a long period of time produces no bony lesion.
- 4 Phosphatase injected in large doses produces only a transient change in the plasma level.
- 5 Phosphatase in excess of the normal is very rapidly destroyed in the body, only a very small amount being excreted.

The significance of these findings, and their bearing on the problem of the high plasma phosphatase in osteitis fibrosa, will be left to the main discussion following the second part of the experimental work.

PART II—EXPERIMENTS WITH PARATHORMONE

The first experiment was undertaken to confirm some results obtained by Jaffe and Bodansky¹⁵ in New York. By injecting quite small doses of parathormone, these workers were able to produce decalcification of the bones of young dogs, of such a degree as to cause deformities and fractures. Histologically, the bones showed many of the appearances of human osteitis fibrosa, such as fibrosis of the marrow, increased osteoclastic activity, and the formation of osteoid tissue, but they were unable to observe any bone cysts or giant-celled tumours. It was decided to repeat this experiment on rabbits and to study in greater detail the histological changes produced in the bones.

Experiment 4—Four adult rabbits were kept on an ordinary diet of greens, oats, and bran, containing an adequate supply of calcium. The calcium, phosphorus, and phosphatase in the blood were determined, and controlled standard radiograms were taken of the bones. They were then each given daily 20 units

of parathormone intramuscularly During the course of injections, further X-rays were taken, and blood chemical examinations were made at intervals, the results of which are shown in *Table VI*

Table VI

DATE AND DOSAGE	RABBIT No	BLOOD		
		Serum Calcium	Serum Phosphorus	Plasma Phosphatase
		Mgmm per 100 c c	Mgmm per 100 c c	Units per c c
May 17	1	14.6	4.6	0.050
	2	12.6	3.5	0.060
	3	11.6	4.4	0.050
	4	15.0	3.5	0.070
20 units of parathormone daily for 14 days				
June 2*	1	14.2	4.8	0.050
	2	13.8	3.6	0.055
	3	14.0	5.0	0.046
	4	13.5	4.2	0.066
Rabbits 3 and 4 40 units of parathormone daily for 14 days				
June 16	3	14.2	3.9	—
	4	13.8	4.0	—
80 units of parathormone daily for 67 days				
June 15	3	12.9	4.7	—
	—	14.1	4.0	—
Aug 22†	3	13.3	4.6	0.070
	4	14.5	3.7	0.075

* Rabbits 1 and 2 killed

† Rabbits 3 and 4 killed

After fourteen days' treatment it was found that no effect was being produced on the serum calcium, phosphorus, and phosphatase, or on the bones, as far as could be determined radiographically. Two of the rabbits were killed and post-mortem examinations made. The bones showed no macroscopic evidence of decalcification, nor were any pathological changes found in the viscera or parathyroid glands. The bones were put in Zenker's fixative for section later, but meanwhile the dosage of parathormone for the remaining two rabbits was increased to 40 units per day. After another fourteen days, blood examinations still revealed no change, so the dose was further increased to 80 units, an amount far in excess of that used by Jaffe and Bodansky. The injections were continued altogether for a period of three months without apparent effect, after which the two remaining rabbits were killed. Post-mortem examination again showed no obvious change in the skeleton or the viscera. The thyroid and parathyroids appeared normal, but in each rabbit a congested lymph-gland was found on each side of the neck. These, together with two of the ribs and a femur, were prepared for section. The lymph-glands showed hæmorrhages and mild inflammatory changes, but the thyroid and parathyroids presented a normal histology. The ribs and femora showed neither decalcification nor increased osteoclastic or osteoblastic activity, and the marrow was unchanged.

The absence of any of the changes reported by Jaffe and Bodansky in puppies given only a small fraction of the amount of parathormone that was given to the adult rabbits in this experiment was striking. It was decided to investigate younger rabbits on a low calcium diet, and to examine not only the serum, but to follow

the excretion of calcium and phosphorus in the urine and faeces as well. In addition, it was arranged to examine the bones histologically during the course of the experiment as well as post mortem.

A consideration of Sherman's¹⁶ food analysis tables showed that the articles of food lowest in calcium, suitable for rabbits, were rice and tomato. In view of the variability of foodstuffs produced in different localities, an analysis was made of these two foods, and also of cabbage and oats in preparation for the third experiment using a high calcium diet. Some differences were found between the results obtained and the figures given by Sherman, both sets of figures are set out in *Table VII*.

Table VII

	CALCIUM grm per 100 grm		PHOSPHORUS grm per 100 grm	
	Author's Result	Sherman's Figures	Author's Result	Sherman's Figures
Rice	0.012	0.009	0.083	0.096
Tomato	0.018	0.011	0.031	0.026
Oats	0.050	0.069	0.296	0.392
Cabbage	0.105	0.106	0.067	0.099

Experiment 5—Two young rabbits 8 weeks old were put on a low calcium diet, consisting of 100 grm of rice and 50 grm of tomato daily. This was equivalent to an intake of 21 mgrm of calcium and 114 mgrm of phosphorus per diem, and a drop of radiostoleum was occasionally added to prevent rachitic changes.

After two months on this diet the animals had put on very little weight and their bones were very thin, but otherwise they appeared to be in an ordinary state of health. They were then transferred to a metabolism cage, constructed so that the faeces remained on the grid which formed the floor of the animal chamber, while the urine passed through and was collected in a receiver underneath the funnel-shaped bottom of the cage. Some difficulty was at first encountered with the zinc from the cage washings in the urine, as this metal tended to interfere with the calcium estimations. The difficulty was overcome by a slight modification in technique, and by coating the interior of the cage with hard wax.

The excreta were divided into four-day periods, and the total weight of faeces and volume of urine for each period were measured. Aliquot parts of faeces and urine were ashed in platinum crucibles, and the ash was extracted with 5 per cent HCl. The calcium in the extract was determined by the oxalate precipitation method, and the phosphorus by the method of Fiske and Subarow¹⁷. In this way the total intake and excretion of calcium and phosphorus could be determined, and the effect of the parathormone on the balance of these elements watched. On the low calcium and phosphorus diet, the balances were both found to be negative, while the serum calcium was 10.8 mgrm per 100 c.c., and the serum phosphorus 3.6 mgrm per 100 c.c. Before injection of parathormone was commenced a portion of rib was resected from each rabbit, as the histological control specimen. The operation was done under local novocain anaesthesia with a cloth over the animal's head, and caused no pain or distress, the rabbit lying quietly without

struggling Ribs were selected for this procedure, partly on account of convenience and ability to repeat the operation a number of times, and partly because Jaffe and Bodansky found the histological changes in the rib much more marked than in the long bones

A course of injections of parathormone was now begun, with increasing doses for each four-day metabolism period, rising from 20 units to 160 units per diem

Table VIII

DAILY DOSAGE OF PARATHORMONE UNITS, 4-DAY PERIODS	CALCIUM			PHOSPHORUS			INTAKE
	Serum	Daily Excretion Urine	Daily Excretion Fæces	Serum	Daily Excretion Urine	Daily Excretion Fæces	
0	Mgrm per 100 c c 10.8	Mgrm 37	Mgrm 27	Mgrm per 100 c c 3.6	Mgrm 130	Mgrm 40	Calcium, 21 mgrm per day
20	—	110	78	—	238	81	
40	12.2	130	13	3.6	155	24	Phosphorus 114 mgrm per day
80	—	77	10	—	123	28	
160	10.3	63	12	—	184	16	

On the eighth day of injection, dosage being now 40 units, a sample of blood was taken and a second rib resected. The blood calcium had risen to 12.2 mgrm, while the phosphorus was still 3.6 mgrm per 100 c c. On the sixteenth day, after

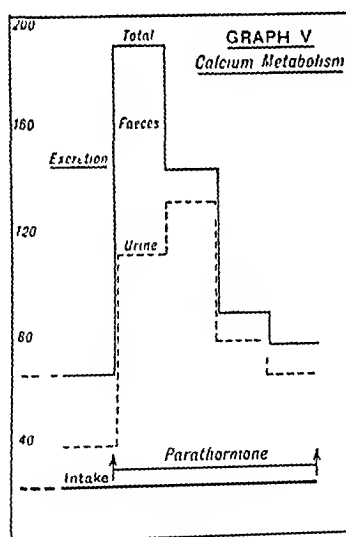


Fig 391

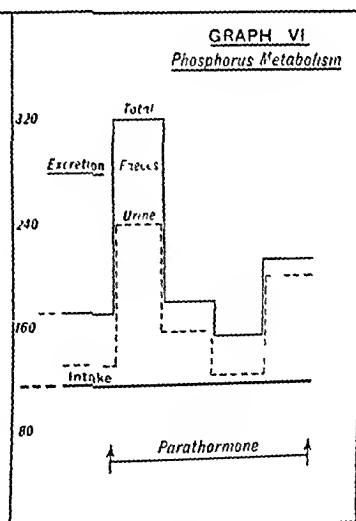


Fig 392

four periods on parathormone, the animals were killed. The serum calcium had returned to below the original figure, in spite of the dose of parathormone having reached by this time 160 units per day. (The last phosphorus estimation was unfortunately spoiled.) As regards the fæces, the metabolism work presented

some difficulty, because on this low calcium and practically non-residue diet the quantity of faeces was very small indeed and variable. The faecal results in the metabolism graphs are therefore somewhat inaccurate, but as the urine excretions are exact, the curves are substantially correct. *Table VIII* shows the figures for the excretion of calcium and phosphorus in the urine and faeces in relation to the intake and serum values of these elements. *Figs 391, 392* are metabolism charts of calcium and phosphorus respectively, constructed from the data in the table.

The results show an extraordinary reaction to the parathormone. The first period before injection shows the continuous negative balance sustained by the rabbit as the result of lack of calcium in the diet, a loss of about 40 mgrm a day. The response to the first dosage of parathormone was immediate, the negative calcium balance being increased to 188 mgrm of calcium lost per day. In the second period, with double the dose of parathormone, the response was less, 143 mgrm of calcium lost per day, in the third period the loss was 87 mgrm and in the fourth 75 mgrm per day. The response of the phosphorus metabolism was even more unusual, only in the first period on parathormone was there an extra excretion of phosphorus. Some compensating mechanism was therefore established against the action of the parathormone, which was sufficiently powerful to overcome the effect of the increasing dosage given. The serum calcium reflects this change, rising slightly after the first two periods, but falling again at the end of the experiment. Post-mortem examination of the rabbits showed no abnormality in the soft parts, including the parathyroid glands. The bones showed the results of the prolonged decalcification, and were very thin and light compared with a normal rabbit. On splitting the femur lengthwise, the cancellous part had fewer and more delicate trabeculae than the control, but the cortical bone in the shaft, although thinner, was hard and compact. The ribs were very frail, but the cortex was a thin, well-defined layer. Nowhere was any abnormality discovered in the marrow.

Histological examination of the ribs removed during the experiment confirmed these post-mortem findings. The first specimen, taken before parathormone injection, showed the effect of calcium deprivation, but the thin cortical layer was composed of laminated bone with well-formed Haversian canals, while active haemogenesis was proceeding in the normal marrow. The young animal, in fact, having been almost deprived of calcium from the age of eight weeks to four months, nevertheless made the best possible use of what it had, and laid down the normal structure of compact bone in the skeleton. The second rib resected showed very little difference from the first, the extra drain of 150 mgrm of calcium daily for eight days having produced no apparent histological change, while microscopic examination of the ribs and femora obtained post mortem showed the same picture. No fibrosis was to be found anywhere in the marrow, no osteoclastic activity, and no new osteoid bone.

In view of the absence of bone effect, and the unaccountable compensation to the parathormone developed by these rabbits, a further experiment was arranged. Jaffe and Bodansky found that the effect of parathormone on the bones of their dogs was much more marked the younger the animal, the most pronounced effect being produced on very young puppies. Also they were able to obtain their effect on the bones irrespective of the amount of calcium in the diet, the only difference

being that the animals on a high calcium diet suffered more severely from the toxic symptoms of hypercalcaemia than did those protected by a low calcium diet. However, as I had so far failed to produce in rabbits anything more than a slight rise in serum calcium, it was decided this time to use very young animals and to give them an adequate diet in all respects, although controlled by the needs of the metabolism work.

Experiment 6—Two young rabbits 6 weeks old, from the same litter, were kept in the metabolism cage on a constant daily diet of 150 gm of cabbage and 75 gm of oats, equivalent to a daily intake of 195 mgrm of calcium and 322 mgrm of phosphorus. The metabolism periods were reduced to three days, and a preliminary period of twelve days on this diet was allowed before the injections of parathormone were begun, in order that equilibrium between intake and excretion should be established. During these four periods the calcium and phosphorus balances, serum concentrations, and plasma phosphatase were determined, standard X-rays were taken, and a portion of rib was resected from each rabbit as before.

Table IX

3-DAY PERIODS	CALCIUM					PHOSPHORUS					PLASMA PHOSPHATASE UNITS PER C C
	Serum	Intake	Excretions			Serum	Intake	Excretions			
			Total	Urine	Faeces			Total	Urine	Faeces	
1	-	535	624	360	264	-	1042	1034	26	1008	
2	-	322	434	283	151	-	738	680	16	664	
3	-	447	498	191	307	-	828	884	16	868	
Rib resected											
4	R ₁ 10 2 R ₂ 12 6	485	464	202	262	4 0 4 1	944	628	13	615	0 18 0 15
Injection of parathormone 40 units daily											
5	-	422	1282	320	963	-	885	649	9	640	
6	R ₁ 13 0 R ₂ 13 6	400	943	341	602	3 7 3 3	865	404	12	392	0 26 0 19
7	-	422	775	222	533	-	885	366	6	360	
8	R ₁ 11 1 R ₂ 12 4	422	682	153	529	3 6 3 6	885	400	8	392	
Rib resected											
9	-	407	614	163	451	-	817	495	17	478	
10	-	422	714	258	465	-	885	404	19	385	
11	R ₁ 10 6 R ₂ 12 8	422	559	141	418	3 9 4 7	885	494	4	490	0 23 -
12	-	404	616	272	344	-	865	350	7	343	
13	-	359	403	180	223	-	596	505	5	500	
Animals killed—ribs removed											

Animals killed—ribs removed

The investigation period then commenced, and the animals were given a constant dose of 40 units of parathormone daily by the intramuscular route. After twelve days, during which the serum changes were followed, another portion of rib was taken from each rabbit for histological examination. During this first part of the experiment, as in *Experiment 5*, the serum calcium of each animal was raised, and there was a marked immediate rise in the amount excreted. During the succeeding twelve days, the excretion of calcium declined progressively, and

the serum calcium returned to its previous level. After two further periods the animals were killed and the third specimen of ribs was obtained post mortem. *Table IX* is a record of the intake and serum values of calcium and phosphorus and the excretions of these in the urine and faeces respectively, together with estimations of the plasma phosphatase. *Fig 393* is a calcium metabolism chart of the experiment with the serum calcium changes of the two animals also shown.

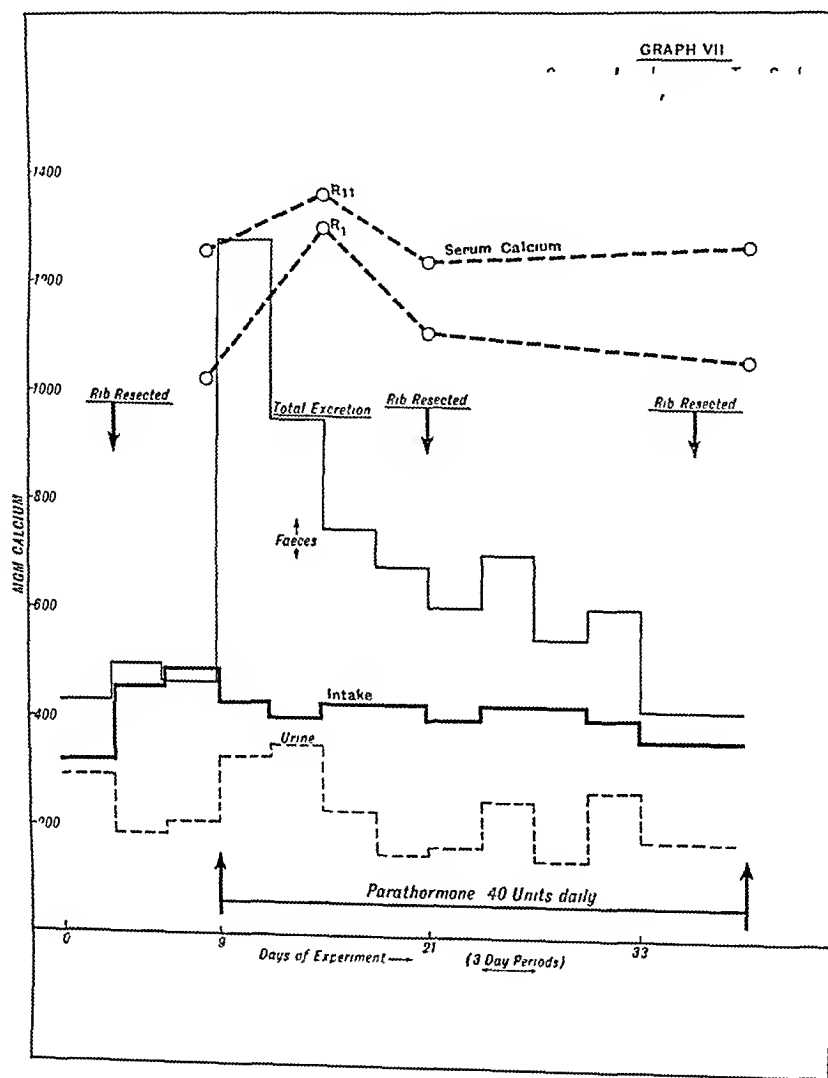


FIG 393

The biochemical results are very similar to those in the preceding experiment and emphasize the following points —

- 1 There is an immediate response to the first injections of parathormone, by a marked increase in the excretion of calcium
- 2 There is a corresponding rise of serum calcium in the early part of the injection period
- 3 The increased excretion of calcium is more by way of the faeces than the urine (unlike human hyperparathyroidism see *Case 2*, p 586)

4 Following the initial excessive excretion, the calcium loss diminishes, *in spite of the continued administration of the parathormone*, until at the end of the twenty-four days the excretion is little more than the intake

5 The phosphorus metabolism responded, if at all, by retention of phosphate rather than increased excretion. This may be associated with the fact mentioned earlier, that rabbits on this type of diet excrete practically no phosphate in the urine (*see Table IX*)

This remarkable result is reflected strikingly in the three specimens of rib. The first specimen (the control) was hard, broke like match-stick, and could be splintered but not cut by a knife. Section of this rib showed a normal compact laminated cortex, with normal red marrow in the centre.

Before the second resection the thorax was palpated and one or two rather plastic areas were felt at the anterior ends of the ribs, in particular in the stump of the rib already resected—at some distance, however, from the cut end.

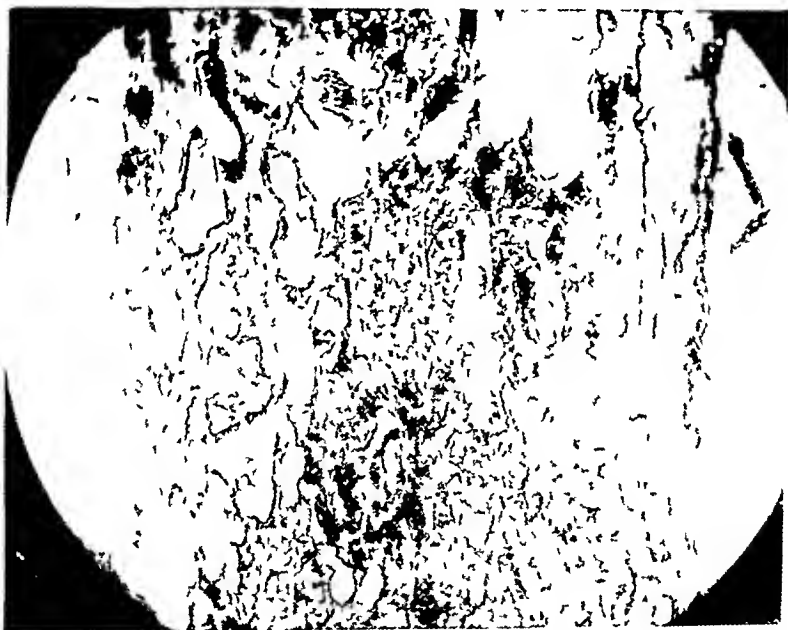


FIG 394—Section of rib showing decalcification by parathormone (Exp 6, First Phase)

This was not corroborated by the radiograms, but respiratory movements rather blurred the outlines of the anterior ends of the ribs. (Also no diminution of density of the shadow or alteration of bone pattern was observed in the long bones.) However, the plastic part of the same rib from which the first portion had been removed was selected for resection, and after removal it was found to be soft and elastic, and could be bent without breaking and cut with a knife. This rib was unfortunately left too long (though only eighteen hours) in the decalcifying fluid, and became somewhat broken up before it could be mounted in paraffin. The section, however, of which a microphotograph is reproduced (*Fig 394*), shows striking decalcification of the cortex, only the distorted connective-tissue framework remaining. The marrow, moreover, shows multiple small hæmorrhages into

its substance, while the line of demarcation between cortex and marrow is irregular and indistinct as compared with Rib 1. It is to be noted particularly that in this rib, removed at the height of decalcification, there is no evidence of osteoclastic activity and no giant cells are present, except those of the hæmatogenic type in the marrow. The marrow hæmorrhages, however, are important in connection with the appearances in Rib 3.

The third specimen of rib was obtained post mortem. When the side of the dissected thorax was viewed by transmitted light, speckly areas were observed in one or two of the ribs, which, however, were not softened. Specimens were taken for section from the speckly areas and from the normal-looking parts. Sections of the latter showed fairly normal appearances, and nothing abnormal was present in the marrow, the cortex was sharply demarcated from the marrow, was well calcified, and contained no giant cells or fibrosis. Sections of the speckly parts of the rib showed striking changes (*Fig 395*), the classical histological appearance



Fig 395—Section of rib (high magnification) during stage of repair of decalcification and marrow hæmorrhage. Note fibrosis of marrow and giant cells—typical osteoclasts. (*Exp 6, Second Phase*)

of osteitis fibrosa. The cortex was partly recalcified, not as a continuous layer of compact bone, but broken up into separate small areas by young fibrous tissue. The marrow was entirely replaced by fibroblasts of the same shape and type seen in sections of von Recklinghausen's disease, and numerous giant cells identical in appearance with those in clinical osteitis fibrosa were present. They were large, rounded cells, with from two to six nuclei in the centre. Some of them lay amongst the fibroblasts which had replaced the marrow, but most lay in apposition to areas of bone. The bone in contact with the giant cells was eaten away in the manner characteristic of osteoclasts, the giant cells lying in typical Howship's lacunæ. Moreover, examination under the high power of the microscope showed that the giant cells presented a different aspect according to their size. The large, fully formed cells, particularly the few lying alone in the young fibrous tissue, had a rounded, oval cytoplasm, with five or six circular nuclei in the middle of the cell

The immature cells, characteristically those forming along an edge of bone, had fewer, more elongated nuclei, which corresponded to processes of cytoplasm radiating from the cell. The appearances were, in fact, that the giant cells were being formed by fusion of two or three of the proliferating connective-tissue cells round about. The stimulus to the fusion seemed to be the presence of an irregular edge of bone amongst the proliferating cells, and all stages from the early fusion to the fully-grown giant cell could be seen.

Other bones of the animals were examined post mortem, but none of these appearances was found. Only one abnormality was discovered—namely, an area of apposition of new bone on the endosteum of the shaft of one of the femora. The new bone had destroyed and replaced part of the marrow, remains of which could be distinguished in the new tissue.

The soft parts of the animals showed no abnormality, there was no evidence of metastatic calcification, and the parathyroid glands appeared normal both macroscopically and on section.

SUMMARY OF RESULTS OF PARATHORMONE EXPERIMENTS

1 Prolonged injection of parathormone into an adult rabbit has no effect on the bones.

2 Rabbits fed from the age of 6 weeks to 4 months on a diet inadequate in calcium have a negative calcium balance, and develop osteoporosis of the bones, which, however, are otherwise normal in structure.

3 Young rabbits given a severe course of treatment with parathormone respond at first by an increased excretion of calcium and a rise in serum calcium.

4 Following this initial response, in spite of continued injection of parathormone, the serum calcium and the excretion of calcium return to normal.

5 Young rabbits on a low calcium diet do not develop bone lesions other than decalcification as the result of parathormone injections, whereas

6 Young rabbits on a high calcium diet do develop local lesions in the bones, similar in histology to human osteitis fibrosa.

7 The lesions in the marrow caused by excess of parathormone are (a) Haemorrhages in the stage of active decalcification, and (b) Fibrosis with giant-cell formation in the stage of healing.

DISCUSSION

The experiments of *Part I* show conclusively that phosphatase given to a rabbit, either over a long period of time, or by high dosage over a shorter period, or by a single massive dose, has no effect whatsoever on the bones. It must be concluded, therefore, that the increased plasma phosphatase observed in decalcifying diseases is the result of the disease process and not the cause of it. Moreover, it has been demonstrated (*Experiment 3*) that phosphatase in the plasma is very rapidly removed from the circulation, a little by excretion, but mainly by destruction. In generalized disease of bone, therefore, with a continuous high plasma phosphatase, if the enzyme is being continually removed, it must also be constantly replaced. This can be explained on the following hypothesis. Bones undergoing prolonged decalcification, as in osteitis fibrosa, are acted upon in two ways. On the one hand is the influence of the decalcifying agent, and on the other the response

of the weakened bone, whereby the osteoblasts are stimulated to form new bone. This they do by an increased output of phosphatase, which, however, is constantly being swept away into the circulation, causing the high concentration of phosphatase observed in the plasma, where, as I have shown, it is destroyed.

The struggle of the bone-cells to compensate in this way the drain of calcium is well illustrated in the mixed histological appearances of a decalcifying bone. The effect of the decalcification is seen in the enlarged Haversian canals and the spaces round the vessels. The reaction of the bone is indicated by the excess of osteoid material and the areas of actual calcification in it, where the local activity of the osteoblasts has concentrated enough phosphatase to cause formation of new fibre bone. This explanation of the high plasma phosphatase is independent of the nature of the decalcifying agent, which is in accordance with the observation that the plasma phosphatase is high in all types of decalcifying diseases, whatever the cause.

This view of the rôle of the osteoblast to form phosphatase is in accordance with a great deal of evidence relating to ossification in other circumstances, both normal and experimental. Many workers, including Leriche and Policard,¹⁸ Jones and Roberts,¹⁹ and others, have observed decalcification in bone in the presence of hyperæmia. According to my view, this is not so much a case of decalcification as of hindrance of recalcification by excessive removal of phosphatase, as the result of hyperæmia. That strain be the stimulus to the formation of phosphatase by the osteoblasts is in agreement also with the view so long held and amply proven by surgeons, that bone is laid down along the lines of strain, resulting, for example, in the mechanically optimum pattern of the trabeculæ in cancellous bone and the hollow compact shafts of the long bones.

Again, the theory of ossification here put forward is not at variance with the idea that the osteoblast is formed by metaplasia of ordinary connective-tissue cells. It merely suggests that the metaplasia consists in the formation of phosphatase by these cells, so that they lay down bone. Huggins²⁰ has shown that other cells which form phosphatase (e.g., bladder mucosa cells), when transplanted into connective tissues, give rise to the formation of ectopic bone. Furthermore, it has been shown by the writer (an unpublished experiment) and others that ligation of the renal artery in a rabbit results in conversion of the kidney, which contains a large amount of phosphatase, into a mass of bone, not an amorphous calcification as occurs in other degenerated tissues. In addition, true ossification has been reported in abdominal scars,²¹ also a site of excessive tissue strain, and the formation of bone was immediately preceded by the appearance of phosphatase in it. It has been observed, too, that the formation of bone in the mesenchyme in the middle of the growing limbs of the foetus—the line of continuous strain—also coincides with the appearance of phosphatase in this situation.

The theory of ossification put forward, therefore, is that connective-tissue cells respond to strain of continuous type by the formation of phosphatase, whereby bone is laid down in the matrix round the cells, which thus become typical osteoblasts.

However, to return to the subject of the high plasma phosphatase, it must not be supposed that this should cause a general deposition of calcium. The calcifying power of the phosphatase in the osteoblast lies in the *local* concentration of its action, whereby the amount of phosphate ions around the cell is increased, and

calcium, which was in equilibrium in the general circulation, is precipitated. A diffuse increase in phosphatase would merely cause a permanent slight shift in the calcium-phosphorus ratio in the tissues, without any precipitation of solid salt.

With regard to parathormone, the absence of any effect on adult rabbits is comparable with a similar result by Bauer²² and his colleagues on adult rats, and by Jaffe and Bodansky²³ on guinea-pigs, while even in dogs the latter workers had difficulty in producing an effect in adults, except with very large doses. But it has been shown that young rabbits do respond to the drug, although temporarily. Indeed, the most striking result of the experimental work is the compensation to the action of parathormone developed by young rabbits, and demonstrated by the changes in serum calcium, calcium excretion, and bone histology. Where, however, the bones have been already subjected to prolonged decalcification, as in *Experiment 2*, the extra drain of calcium due to the parathormone seems to be distributed evenly over the skeleton, and there are no areas of severe damage as in *Experiment 3*. This may be explained by the protection afforded by the low calcium diet (*see below*) or perhaps by the somewhat greater age of the rabbits in the former experiment.

Jaffe and Bodansky found that the effect of parathormone was more pronounced the younger the dog, an observation that is in agreement with mine, but they were able to produce a relatively permanent effect on the bones of puppies, with only a small fraction (2 to 5 units) of the parathormone given to my rabbits. Moreover, the effect was a continuous one, although it diminished in intensity as the dog grew older, whereas in rabbits the effect was very transient. It was, in fact, remarkable how rapidly the affected parts of the ribs became first decalcified and then recalcified (albeit with other changes) in the space of one month.

The nature of the compensatory process is not clear. Pugsley²⁴ reported a similar compensation in rats and was able to show that it was not due to an immune body in the serum, for inoculation of an untreated rat with serum from an 'immune' rat did not protect it from the parathormone.

It is possible that man also may have a mechanism of compensation to parathormone, although slight compared with the rabbit, as it has been noticed that the curative effect of parathormone in cases of tetany following parathyroidectomy diminishes steadily the longer it is used. Apart from the question of compensation, there is a metabolic difference between the human being and the rabbit in the response to parathormone, for, as these experiments show, the resulting increased excretion of calcium occurs by way of the faeces in the rabbit, whereas in man the excess is eliminated almost entirely by way of the urine (*Case 2*, p. 586). It has been shown that the rabbit excretes no phosphate in the urine on what is, for this animal, a normal diet (cabbage and oats), whether given parathormone or not. But Albright, Bauer, and Aub have shown that the decalcifying action of parathormone in ordinary doses in man is the result of excessive phosphate excretion in the urine. It seems probable, therefore, that the compensation which occurs in rabbits, but not human beings, is connected with this difference in phosphate metabolism in the two species.

However, in spite of the compensation, it is quite clear that parathormone will produce localized lesions of osteitis fibrosa in the ribs of rabbits. Moreover, it seems that the question of how the lesion developed can be answered by these experiments, particularly *Experiment 6*.

This experiment is distinctly divisible into two phases—decalcification and compensation. The decalcification in the first phase is the direct effect of the parathormone. It is localized in type, affecting especially the ribs at their sternal ends, and is accompanied by hæmorrhages into the marrow. The cause of the hæmorrhage is probably as follows. It has been shown that hypercalcæmia is toxic to body cells, and that in high doses parathormone causes a rapid degeneration of the viscera. Now the concentration of calcium in the blood of the capillary vessels at the actual site of decalcification, must be much greater than that in the general circulation. Consequently, these vessels are subject to the most toxic action of the calcium, with the result that the endothelium of the bone capillaries is damaged and extravasation of blood takes place into the marrow. The dependence of the hæmorrhage on hypercalcæmia in this way would also explain the absence of hæmorrhages in the rabbits on a low calcium diet, as those animals had a correspondingly lower serum calcium than those on an adequate calcium diet. In the latter animals the extra concentration of calcium in the bone capillaries, added to the general high serum calcium, was sufficient to cause the hæmorrhages into the marrow.

It seems, therefore, that marrow hæmorrhages depend on the severity of the decalcification, whereby calcium is concentrated in the vessels of the bone to such a degree as to damage the vascular endothelium.

To return to the second phase of my experiment, it was found that where there had been no damage to the marrow by hæmorrhage, the bone recalcified normally without alteration of structure, as in *Experiment 5*. Where, however, the marrow had been damaged by hæmorrhage it was replaced by a proliferation of connective-tissue cells, the hæmopoietic elements being removed by phagocytosis. It was at this stage, and not before, that the giant cells made their appearance. Moreover, the giant cells appeared to be formed *in situ* from the proliferating (phagocytic) cells, and were then indistinguishable from ordinary osteoclasts. Indeed, the formation of giant cells seemed to be stimulated by the presence of irregular bone. If it be accepted that these giant cells are essentially the same as ordinary osteoclasts, the absence of osteoclasts in the stage of decalcification is significant, for it indicates that osteoclasts take no part in decalcification by parathormone. The giant cells, however, appeared when there was real osteoclastic work to do in remoulding the damaged bone to the normal structure. The irregular bone laid down among the fibrous tissue was mechanically useless to the strength of the bone, and it was these islands of bone that the giant cells were absorbing. Here, then, is an apparent distinction between osteoclastic activity and the removal of calcium for metabolic purposes. In metabolic decalcification too slow to cause damage to the bone capillaries, the histological picture is one of simple solution of lime salts—halisteresis—with large spaces round the vessels, but no osteoclasts. If, however, phagocytic activity is aroused by marrow damage, in a disease like osteitis fibrosa with irregular deposition of bone, the local phagocytes fuse together to remove the unwanted fragments.

This conception of the giant cells as a part of the cellular reaction to the hæmorrhage is in accordance with the views of Kolodny,²⁵ Codman, and Mallory. The first quotes experiments by Konjetsny, who produced marrow hæmorrhages by mechanical traumata and found that they healed either by connective-tissue-cell proliferation, with giant cells of this type, or that small cysts were formed. Kolodny then supposes that, their phagocytic function complete, the giant cells remain

amongst the connective tissue and form the giant-celled tumours of von Recklinghausen's disease or disappear and leave cysts. This view of the disease is in accordance with many of its clinical characters—for example, the age of onset and the distribution of the lesions. The hæmorrhages would naturally occur when and where the bones are most vascular, and so the disease typically arises in young people, and, except in advanced cases, the cysts and tumours which follow the hæmorrhages are mainly at the ends of the bones. When it is also remembered that von Schupp²⁶ has reported cases of true hyperparathyroid decalcification without giant-cell tumours or cysts, it seems clear that these are due, not specifically to parathormone, but to any agent that will decalcify sufficiently rapidly to cause vascular damage in the bone and hæmorrhages into the marrow.

If one may review the position of von Recklinghausen's disease in the light of these findings, there are the following points to consider —

1 The hypercalcæmia is definitely due to hyperparathyroidism, and to no other cause

2 The low serum phosphorus is an effect of ionic dissociation, and is not specific to parathormone

3 The high plasma phosphatase is indicative of the reaction of the osteoblasts to the decalcification, and is not specific

4 The excessive excretion of calcium in the urine, although not specific to hyperparathyroidism (*see Case 4*, p 588), is strongly suggestive of it

5 The histological appearances—the extreme decalcification, marrow fibrosis, cysts, and giant-celled tumours—are indicative of a powerful decalcifying agent. Hyperparathyroidism is the most likely condition to produce them, although other causes are possible

A typical case of hyperparathyroidism, therefore, presents all the above characters, which, taken together, establish the diagnosis, but only one of these is itself pathognomonic—the absolute hypercalcæmia

SUMMARY OF CONCLUSIONS

1 The high plasma phosphatase in decalcifying disease is indicative of the extent of the bony reaction

2 A suggestion is put forward with regard to the mechanism of the physiological process of ossification

3 A distinction has been suggested between metabolic decalcification and the moulding of the bones by osteoclasts

4 Differences in phosphate metabolism between rabbits and human beings have been indicated to account for—

5 The compensation established in rabbits to the effect of parathormone

6 Areas of bone showing the classical histological features of osteitis fibrosa have been produced in rabbits by parathormone

7 These features have been shown to be the result of the healing of marrow hæmorrhages and are not necessarily specific to the action of parathormone

8 The giant cells have been shown to arise *in situ* by fusion of proliferating connective-tissue cells

9 The giant cells so formed are indistinguishable from osteoclasts

APPENDIX

During the course of this work, various clinical cases of abnormal calcium metabolism in the wards of St Bartholomew's Hospital were investigated. A few of these are appended to illustrate some of the statements made in this paper. Attention was concentrated on the serum calcium and phosphorus, and plasma phosphatase, and their relation to the excretion of calcium. The technique of the calcium and phosphorus balance experiments was as follows —

The patient was kept in bed on a fixed diet, low in calcium, but with a sufficient calorie value for the requirements of basal metabolism. The proportion of foodstuffs was adjusted to give a balance between acid- and alkali-forming foods in order to avoid the effects of acidosis. At the end of eight days of this diet, the intake and output of calcium and phosphorus had become steady, and the experimental period proper commenced and continued for a fortnight. The urine was collected in forty-eight-hour specimens, and the faeces in four-day specimens, marked off by doses of carmine by mouth. The excreta were measured, and aliquot parts analysed by the method described above in the work on rabbits.

The results of the analysis of four cases are here reproduced—cases of renal rickets, of typical hyperparathyroid osteitis fibrosa, before and after removal of a parathyroid adenoma, a case of non-parathyroid osteitis fibrosa, and one of early Paget's disease.

CASE REPORTS

Case 1—Renal rickets

The case of renal rickets referred to in the introduction was a typical one. It was a youth of 19 with advanced chronic parenchymatous nephritis, and a blood-urea of 80 mgrm per 100 c.c., which later rose to nearly 400 mgrm. This boy had marked decalcification of all bones, but no deformities. The plasma contained 0.97 units of phosphatase per cubic centimetre, about ten times normal—indicative of a pronounced bony reaction.

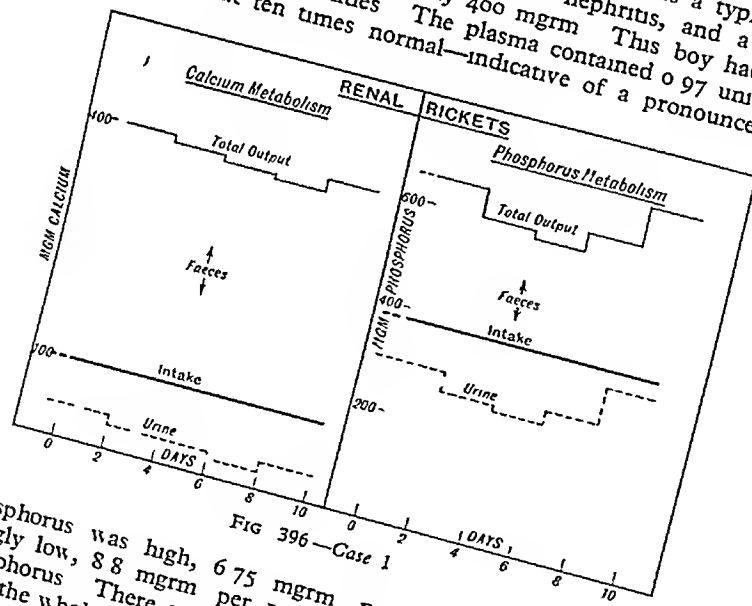


Fig 396—Case 1

The serum phosphorus was high, 6.75 mgrm per 100 c.c., and the serum calcium was correspondingly low, 8.8 mgrm per 100 c.c. Fig 396 illustrates the metabolism of calcium and phosphorus. There is a marked excretion of calcium, about four times the intake, and almost the whole of it is eliminated by the bowel and very little by the damaged kidneys. The excretion of phosphorus, also by the colon, is greater than the intake in spite

of the phosphate retention by the kidney. The mechanism of the whole process is (1) Phosphate retention by the kidney, (2) Elevation of serum phosphorus, with depression of serum calcium, (3) Excretion of the excess phosphorus by the bowel as calcium phosphate, and (4) Mobilization of further calcium phosphate from the bones to make up the loss. In this way a continuous process of decalcification is established.

Case 2—Hyperparathyroidism

This was a typical case of hyperparathyroidism of long standing. The serum calcium was 14.7 mgrm per 100 c.c. and the serum phosphorus was 1.47 mgrm per 100 c.c., which illustrate the absolute hypercalcaemia and the consequent depression of the serum phosphate, in accordance with the law of ionic dissociation. Decalcification with giant-celled tumours

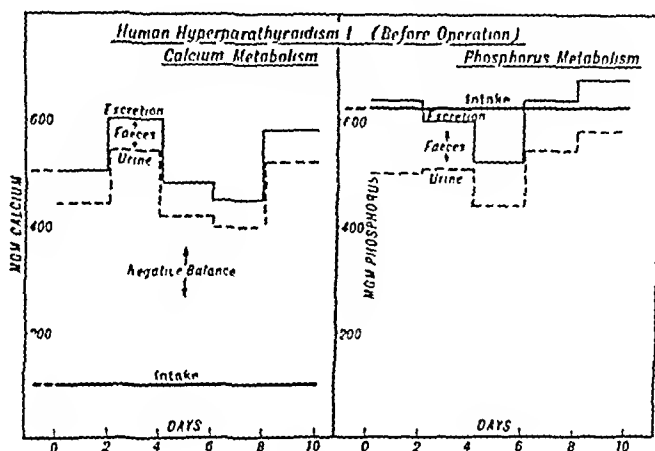


FIG 397—Case 2

and cysts in the bones had been treated for years, and the patient had multiple deformities. The plasma phosphatase was raised to 0.33 units per cubic centimetre, only about four times normal, indicating that the resistance of the osteoblasts was at a low ebb. In the metabolism graphs (Fig 397) it will be seen that there was a negative calcium balance of 350 mgrm of

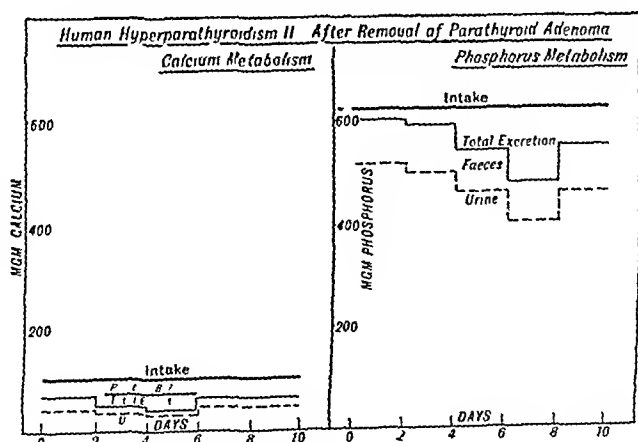


FIG 398—Case 2

calcium per diem. The phosphate metabolism does not show any marked abnormality. The curves are characteristic of human hyperparathyroidism, in that the excretion of calcium and phosphorus is almost completely by way of the urine, differing in this respect from rabbits.

After operation the serum calcium fell immediately to 8.4 mgrm, and the serum phosphorus fell to 2 mgrm, rising during the next fortnight to 2.9 mgrm. At this time mild symptoms of tetany were observed, which, however, quickly disappeared. Five months after operation the patient's metabolism was again investigated on the same diet as previously. The serum calcium had risen to 11.2 mgrm and the phosphorus to 4.4 mgrm per 100 c.c., while the plasma phosphatase was 0.9 units per cubic centimetre—all figures

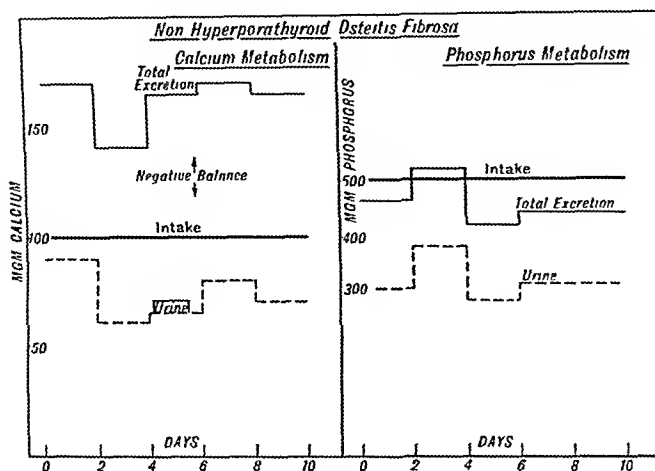


FIG 399—Case 3

within normal limits. A striking change will be seen in the metabolism graphs (Fig 398), the previously negative calcium balance having become a positive one of 40 mgrm of calcium laid down each day in the bones. The phosphate metabolism charts are little altered.

Case 3—Non-parathyroid osteitis fibrosa

In contrast with the preceding case, this was one of non-parathyroid osteitis fibrosa, established by histological examination of the bones. Serum examination, however, showed

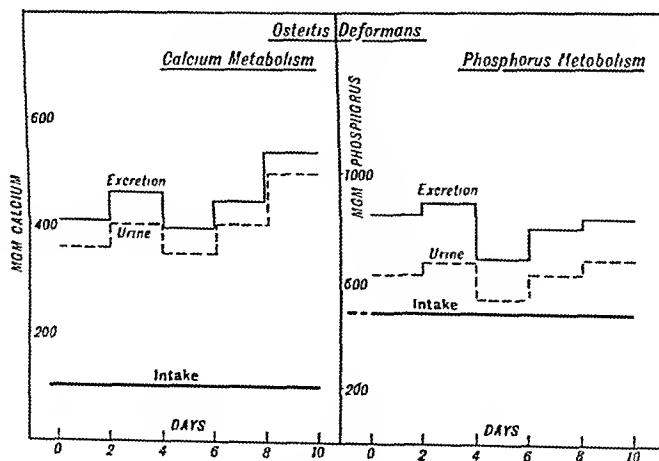


FIG 400—Case 4

a low calcium, 8 mgrm per 100 c.c., and phosphorus 2.6 mgrm per 100 c.c., evidence of diminution of parathyroid activity rather than the reverse, perhaps in compensation for the other agent at work which was decalcifying the bones. The bony response was again not great, indicated by the plasma phosphatase of 0.21 units per cubic centimetre. The disease was of the multiple localized type, about half the bones of the body being affected. The

metabolism graphs (Fig 399) indicate a slight negative calcium balance, the excretion, however, being rather more in the faeces than the urine, in contradistinction to the hyperparathyroid case. The cause of the decalcification in this case was not discovered.

Case 4—Early Paget's disease

The patient was a man of 49 admitted as the result of bilateral coxa vara, who had noticed symptoms for only eighteen months. There was diffuse decalcification of the femora, tibia, and humeri, but no bone cysts or tumours. The serum calcium was 11.5 mgrm per 100 c.c. and the phosphorus 3.6 mgrm per 100 c.c. The metabolism graphs (Fig 400) show a striking resemblance to those of hyperparathyroidism, the marked negative balances of calcium and phosphorus occurring in each case by way of the urine. Such a graph is unusual in Paget's disease, but the disorder consists of a period of decalcification, of which this case is an example, followed by compensation and sclerosis of bone, with corresponding variations in the metabolism figures.

I should like to express my thanks to Professor Kettle, Dr Harrison, Dr Archer, and Miss K. Hare for valuable help with technical points in this work and for permission to use the facilities of the Pathological Department of St Bartholomew's Hospital. I wish to thank also Professor Gask, Professor Fraser, Mr R. C. Elmslie, and Mr G. L. Keynes for their interest and for permission to investigate their cases, and Mr Paterson Ross, to whom I looked for inspiration on this subject.

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REFERENCES

- ¹ TELFER, S. V., *Quart Jour Med*, 1922-3
- ² PETERS and LISERSON, *Jour Biol Chem*, 1929, lxxiv, 155
- ³ GREENWALD and GROSS, *Ibid*, 1925, lvi, 185
- ⁴ PETERS and VAN SLYKE, *Quart Clin Chem*, 811
- ⁵ ROBISON, *Biochem Jour*, 1924, xviii, 1161
- ⁶ COLLIP, *Jour Biol Chem*, 1925, lxi, 101
- ⁷ COLLIP, BAUER, ROPES, and AUB, *Jour Clin Invest*, 1929, vii, 139
- ⁸ ALBRIGHT, BAUER, ROPES, and AUB, *Jour Exper Med*, 1931, lxi, 591
- ⁹ BARR and BULGER, *Amer Jour Med Sci*, 1930, cxliii, 449
- ¹⁰ HUNTER, D., *Lancet*, 1931, April
- ¹¹ SELYE, *Arch of Pathol*, 1932, iv, 60
- ¹² MCJUNKIN et al., *Ibid*, 1932, iv, 60
- ¹³ KAY, H. D., *Jour Biol Chem*, 1930, lxxv, 183
- ¹⁴ BOURNE, M. C., *Biochem Jour*, 1932, xvi, 1
- ¹⁵ JAFFE and BODANSKY, *Jour of Exper Med*, 1929, vii, 139
- ¹⁶ SHERMAN, *Chemistry of Food and Nutrition*
- ¹⁷ FISKE and POLICARD, *Jour Biol Chem*, 1925, lvi, 387
- ¹⁸ LERICHE and SUBARROW, *Physiologie et Pathologie de l'Os*, 1927, Paris
- ¹⁹ JONES and ROBERTS, *Brit Jour Surg*, 1934, Jan
- ²⁰ HUGGINS, *Arch of Surg*, 1931, xlii, 377
- ²¹ HUGGINS, *Biochem Jour*, 1931, xlv, 728
- ²² BAUER, AUB, and ALBRIGHT, *Jour of Exper Med*, 1929, xlv, 145
- ²³ JAFFE and BODANSKY, *Jour Biol Chem*, 1930, lxxxviii, 629
- ²⁴ PUGSLEY, *Jour Physiol*, 1932, lxxii
- ²⁵ KOLODNY, *Surg Gynecol and Obst*, 1927, xlv, 1
- ²⁶ VON SCHUPP, *Deut Zeits f Chir*, 1931, ccxxiii, 195

ETIOLOGY OF TRAUMATIC SHOCK

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INTRODUCTION

THE definition of traumatic shock has always caused difficulty to writers on the subject, and this difficulty has often been overcome by abandoning the attempt and providing instead a clinical picture of the syndrome. The type of traumatic shock with which this paper is solely concerned is frequent in civil practice. A very typical case was observed early in the course of the investigation. A 6-year-old boy was run over by a bus—the vehicle passed over both thighs—and he was admitted to hospital in a state of shock. In this state he died some twenty-four hours later. At autopsy there was much extravasation of blood into the muscles around the fractured ends of the femora, and this extravasation extended into the retroperitoneal tissues. There was intense pallor of the skin. The omentum, intestines, liver, and kidneys were also pale in colour, and on section of these organs the cut surfaces did not bleed. There was no evidence of congestion of the splanchnic vessels, nor were any petechial hæmorrhages observed. The lungs were pale and the heart was small, contracted, and only moderately full of blood. No vital organ had suffered injury, and the case was in every respect similar to one that prompted Crile, then still a student, to direct his energies towards a solution of the problem of shock. It must be remembered that whatever the ultimate explanation of shock, it was the application of this experimental work which enabled Crile to lay down the important principle of shielding the patient from all nociceptive impulses, whether physical or psychological. The same type of shock was produced experimentally in the cat in the classical experiments of Cannon and Bayliss, and their method has been used in the present series of experiments.

Much work has been done on the shock which may result from trauma to or even handling of the intestines and on the collapse which attends extensive burns of the skin, and efforts have been made to provide an explanation for shock which will also explain the fatal collapse of intestinal obstruction or general peritonitis. None of these conditions will be considered here, and any conclusions reached apply solely to traumatic shock of the type just described.

Although, as the literature amply shows, the syndrome of traumatic shock had always attracted the attention of English surgeons—the term was adopted almost in its literal form in France and in Germany—the problem assumed such proportions during the Great War that a new theory arose to explain the syndrome, it

might be truer to say that a very ancient theory was revived (*Lancet*, 1934, 1, 1016). The importance of nervous impulses and of fluid loss into the traumatized area had always been recognized, but in the experiments carried out by Cannon it seemed to him that neither of these factors was adequate to account for the collapse and death which followed trauma inflicted on the thigh of the etherized cat. In 1910 Dale and Laidlaw had described the action of histamine on the experimental animal. Dale resumed this work with Richards and Laidlaw early in the war on account of the possible interest by analogy of the shock-like condition which large doses produced. Although the actual production of histamine from fresh animal tissues was only to be established beyond doubt by Best, Dale, Dudley, and Thorpe in 1926, Cannon was attracted by the possibility that this or some similar product was absorbed from the traumatized area, and some of his experiments lent support to this view. Although Dale, Laidlaw, and Richards were careful to point out the important respects in which the circulatory collapse of histamine poisoning differed from that following on trauma, there was a tendency for the toxic factor not merely to be granted a certain hypothetical importance but for it to be regarded as the main and most important agent in the production of the syndrome. To a large extent this tendency has persisted, at least in clinical practice, despite the volume of experimental evidence which has accumulated in the years following the war against the toxic theory of shock. Dale has recently pointed out that "many details of the work done in war have needed revision in the light of investigations made under the unhampered and unhurried conditions of more normal times."

The confusion which surrounds this problem sufficiently justifies a re-examination of the experimental issues as a preliminary to some new therapeutic measures which are under consideration. That a need exists for an improved treatment of traumatic shock requires little emphasis. McDowall, in a recent Arris and Gale Lecture, pointed out the benefits which should result from a more systematic study of the results of trauma. From the experimental side there is abundant proof of the inefficacy of intravenous infusions in the treatment of established shock. The work of Padgett and Orr disposes as effectively of one of the newer methods of infusion—the use of a solution of glucose combined with injection of insulin—as similar experiments, like those of Smith, disposed of the older type of infusion consisting of gum saline or normal saline. The case against the treatment of traumatic shock by intravenous infusions and the evidence against the toxic theory of shock were summarized by one of us (L. O'S.) in 1931, and from our clinical observation we would endorse the opinion recently expressed by Blalock that there is at present no satisfactory treatment for shock which has persisted for several hours. It has been our experience that the subject of severe trauma who does not show some signs of recovery under established modes of treatment within two or three hours of his injury is almost inevitably doomed.

It seemed clear that a therapy directed towards three possible factors—the existence of one, the toxic factor, being at any rate doubtful—was less likely to be effective than a therapy directed against two certain factors. As a corollary to this an attempt to assess the relative value of the two remaining factors has also been made. Although a final judgement has not yet been reached, enough evidence has accumulated to show that the fluid-loss factor does not occupy the position of almost exclusive importance which has been assigned to it by some recent workers.

Feldberg and Schilf have recently stated that none of the animal experiments so far published have sufficed to establish the truth of the toxic theory of shock. We would go further and state that, so far as the type of traumatic shock here considered is concerned, the animal experiments published, the experiments recorded in this paper, and such observations as have been made on the blood of traumatized patients quite definitely disprove the theory.

Two sets of experiments are quoted by Feldberg and Schilf as providing suggestive, although not final, evidence in favour of the toxic theory. The first consists of the experiments of Voegtlin and Dyer, who noted that rats—notoriously resistant to histamine (M L D 900 mgrm per kilo)—were also resistant to trauma. Ten rats were subjected to trauma and all recovered. There are no records of blood-pressure during and after trauma. It is difficult to assess the degree of trauma inflicted, for only by careful post-mortem examination and weighing of a traumatized extremity can the degree of trauma inflicted during life be ascertained. Once histamine as a cause of traumatic shock following trauma to the thigh can be excluded, the special idiosyncrasy of the rat towards histamine bears no more relation to the problem than do any other of his idiosyncrasies, of which there are doubtless many. The second experiments quoted are those of McIver and Haggard, who constructed an ingenious animal preparation by means of which the amputated lower extremities of one cat were supplied with blood from a second cat through an artificial vascular anastomosis. Trauma to the amputated extremities was followed by a fall in blood-pressure of the donor cat. Since it was quite impossible for nervous impulses to reach this animal, it was assumed that the fall in pressure could only be due to the passage of some toxic substance from the traumatized area. Again there is no record of the post-mortem appearances or weight of the traumatized limb, so that the factor of local fluid loss into the traumatized area is not controlled. We repeated one experiment of this type, and the fluid loss, although smaller than is usual following our method of trauma, seemed adequate to account for the fall in pressure obtained, because the setting up of the preparation involved a certain amount of incidental trauma and blood loss. We have also found it possible by means of a less elaborate preparation to isolate the lower limb from the general circulation and to establish the fact that, even when the venous ligatures carried out are adequate to prevent the absorption of histamine injected into the limb, trauma—provided the arterial supply is intact—leads to a typical onset of shock.

A complete resume of the evidence against the toxic theory of shock which has been published in America and most European countries during the last ten years will not be attempted. The contributions of such experienced surgeons as Rehn and Coenen may be mentioned as having had an important influence on European opinion in this respect. A rather rare international agreement on a controversial issue is illustrated by a short list of workers who, for one reason or another, have decided against the toxic theory of shock—Parsons and Phemister (America), Bolten (Holland), Kalalova (Czechoslovakia), Ewig and Klotz (Germany), Simonart (Belgium)—and further search could enlarge this list almost indefinitely.

The publications which have a direct bearing on the work presented here are those of Blalock and his associates, of Parsons and Phemister, of Friedlander and Lenhart, of Schneider, Simonart, and Smith.

To Blalock we are indebted for the discovery of a satisfactory technique for

weighing the traumatized extremity. In the earlier experiments amputation was done through the thigh, and the negligible difference in weight between the normal and the traumatized extremity led Cannon to believe that the factor of local hæmorrhage could not be of importance. Blalock observed that the hæmorrhage and exudation of fluid which results after severe trauma to the thigh extends upwards into the muscles of the flank (we also observed this phenomenon in a fatal case of trauma of the thigh in man), and he found that if hindquarter amputation was carried out, a very significant difference in weight between the normal and the traumatized side could be demonstrated. Blalock also found that shock still developed when a limb was traumatized after a tourniquet had been applied so as to obstruct all vascular channels with the exception of the femoral artery. There was no path for absorption of any toxin, and Blalock attributed the shock to hæmorrhage into the traumatized area. Trauma to an extremity after occlusion of both arteries and veins was not followed by the development of shock, and this Blalock explained by the fact that under these conditions hæmorrhage into the traumatized area was impossible. Blalock also demonstrated that the infusion of blood from a shocked animal into a normal dog produced a rise in blood-pressure—not a fall as would be expected if a depressor substance were present in the circulation of the shocked animal. Thus Blalock failed to confirm the view of Cannon and Bayliss as to the toxic origin of traumatic shock, but he is prepared to accept the other main conclusion of these workers on the relatively unimportant part played by the nervous system. He himself has not published any observations on the effect of nerve section on the onset of traumatic shock, but he states that trauma carried out after a spinal anæsthetic still produces characteristic effects. Details of these experiments have not been published.

Parsons and Phemister used seventy dogs in their series of experiments, and in most cases traumatized one thigh in the manner of Cannon and Bayliss. They were able to show that the blood from a traumatized extremity when injected into a normal animal failed to lower the blood-pressure. Another significant finding was that traumatized muscle from an animal which had developed shock, when removed from the body and treated by biochemical methods, gave as high a yield of vasodilator substances as did an equivalent weight of normal muscle. Clearly, if the shocked state had been even in part due to absorption of vasodilator substances from the traumatized area, a lower yield should have been obtained. It was found that a dog whose blood-pressure had been reduced to 60 mm of Hg by the injection of histamine was remarkably resistant to hæmorrhage. In order to produce death in such an animal some 600 c.c. of blood had to be removed, while in a dog of similar weight but with a low blood-pressure following trauma 100 to 200 c.c. of blood constituted a fatal hæmorrhage. Shock due to a local production of some vasodepressor substances in the traumatized area should be averted by ligation of the main veins draining the area, but Parsons and Phemister found that this intervention hastened rather than retarded the onset of shock after trauma to an extremity. It is stated that concentration of the blood is a constant feature in histamine intoxication, but in none of their experiments was any change other than dilution of the blood observed. Parsons and Phemister also recorded that the blood recovered from the traumatized area showed no tendency to clot even if kept for some days. No explanation was found for this fact, although attempts were made to relate it to an increased production of heparin as a response

to the trauma. This finding may possibly explain the comparative rarity of a blood-clot forming in the carotid cannula in our traumatized animals and the extreme frequency of such a complication in our histaminized animals.

Freedlander and Lenhart carried out experiments on trauma to the hinder extremity of the cat. They too found that concentration of the blood was not a feature of this type of experimental shock, although marked concentration occurred in the early stages of ether anaesthesia. They confirmed the view of Blalock that the important factor in traumatic shock was the local loss of fluid into the traumatized area. They conclude that the nervous factor is unimportant, and certainly some of the animals subjected to a preliminary section of the spinal cord died very soon after the infliction of trauma. At the same time it is significant that one animal which had been subjected to section of the spinal cord and excision of the lumbar sympathetic chain some days prior to trauma still maintained a blood-pressure of 118 mm. of Hg (initial level was 120 mm. of Hg) when the experiment was terminated some two hours later. At this period after trauma all the control animals were either dead or in a state of shock.

Simonart carried out some experiments on the cat in which the hind leg was subjected to trauma in the usual way. He concluded that there was no evidence for a toxic factor in the production of traumatic shock. In contradistinction to most other recent workers, Simonart considers that the nervous system plays a decisive rôle. He has carefully examined the original tracing on which Cannon based his view as to the relative unimportance of the nervous system under the conditions of experimental trauma (*Traumatic Shock*, p. 146). The tracing shows the blood-pressure of a cat subjected to trauma after section of the femoral and sciatic nerves. Simonart points out that 1 hour 40 minutes after trauma the blood-pressure was still 88 mm. of Hg, while in the control experiments a much lower level had always been reached in a similar space of time. Simonart records an experiment in which he subjected the limb to trauma after section of the femoral and sciatic nerves and occlusion of the femoral artery and vein. There was no significant fall in blood-pressure on releasing the vessels, while a similar intervention on a limb with an intact innervation led to a progressive fall in blood-pressure. In his opinion an intact nerve-supply is essential for the production of traumatic shock.

In 1928 M. I. Smith published a careful series of animal experiments on the subject of traumatic shock. He found that the blood removed from the femoral vein of a traumatized extremity when infused into a normal animal failed to produce a vasodilator effect. On the basis of this and some other experiments he decides that, whatever the mechanism of traumatic shock, local toxin production plays no part in the syndrome. While recognizing the importance of local fluid loss, Smith comes to the final conclusion that a depression of the peripheral vasomotor mechanism is the essential feature in traumatic shock—a return to the Meltzer inhibition theory of some thirty years ago.

Holt and Macdonald have recently repeated and confirmed some of the experiments of Blalock and of Smith. One point in their technique is at variance with our own findings. Ligature of the iliac vein—at least in the cat—does not prevent the absorption of histamine injected into the limb. A very complex series of ligatures is required in order to separate the vascular system of the lower limb from the general circulation, as will be demonstrated in the experimental part of this

paper These workers accept the original experiments of Cannon and the later experiments of Freedlander and Lenhart as sufficient evidence against the importance of the nervous factor They found in their own experiments that spinal anaesthesia failed to prevent *initial* fall in blood-pressure on traumatizing an extremity—there is no note as to the course run by these animals as compared with a normal control series In short, while prepared to abandon the toxic factor in shock together with Blalock, they also follow him in his view as to the relative unimportance of the nervous factor

Despite the frequent reference to the toxæmia present in cases of shock in man, it has been impossible to discover records of any experiment in which the administration of blood from such a patient to an animal has produced vasodilator effects It is perhaps significant that when such experiments were carried out by Schneider using the blood of twenty patients, some of whom had suffered extensive burns and others operative trauma, in no case was the presence of a vasodepressor substance demonstrated

One assumption in the hypothesis that attributes the symptom-complex of traumatic shock to the elaboration of vasodilator toxins in the area of trauma is that the amount of these depressor substances obtainable from the traumatized area must clearly be adequate in quantity to produce shock The quantity of depressor substances which can be extracted from the tissue to be subjected to trauma must be of a quantitative order at least equal to the minimum dose necessary to produce the phenomena of shock

Histamine and other vasodilator substances are readily extracted from most tissues Lung and liver and some other tissues have a high content of these toxins, especially histamine The amount prepared from muscle is very low even by the most careful biochemical extraction, Thorpe showed that extracts of voluntary muscle of the cat have a depressor activity equivalent to 1 mgrm of histamine per kilo of fresh tissue The weight of the entire musculature of the thigh, only a small proportion of which is actually lacerated in our experiments, constitutes about 5 per cent of the total weight of the animal Dale and Laidlaw have shown that while small doses of histamine, 0.01 to 0.1 mgrm, produced transitory and evanescent effects on blood-pressure, a dose of 1 to 2 mgrm per kilo of body weight, injected intravenously, will produce a typical shock-like failure of the circulation Thus the dose of histamine necessary to produce shock in a 4-kilo cat by the intravenous route is 4 to 8 mgrm The total amount of depressor substances that can be obtained by thorough trituration and careful extraction from the 200 mgrm of thigh muscle is only of the order equivalent to 0.2 mgrm of histamine These extracts, prepared biochemically in the laboratory from the entire muscle mass of the thigh, are in no way comparable to the much smaller amount of laceration of the thigh muscles as seen in the trauma adopted in these experiments Dale has also shown that relatively large amounts of histamine can be tolerated by the cat when administered by slow intravenous infusion In a series of experiments we have been able to demonstrate that the amount of histamine necessary to produce shock by intramuscular injection is ten times the amount necessary by the intravenous route (*Fig 401*) In our experiments the hypothetical depressor substances produced as a result of trauma are presumably elaborated in the muscles We may also presume that they are absorbed slowly into the circulation Thus it becomes sufficiently clear that the total amount of toxins which could possibly be

freed for absorption, under the most favourable conditions, from the entire musculature of the thigh would prove inadequate to produce shock Weiss, Robb, and Ellis have demonstrated that as much as 20 mgrm of histamine per hour may be infused into the venous system of a man *without* producing any depressor effect on blood-pressure Probably this represents the utmost amount of depressor substances

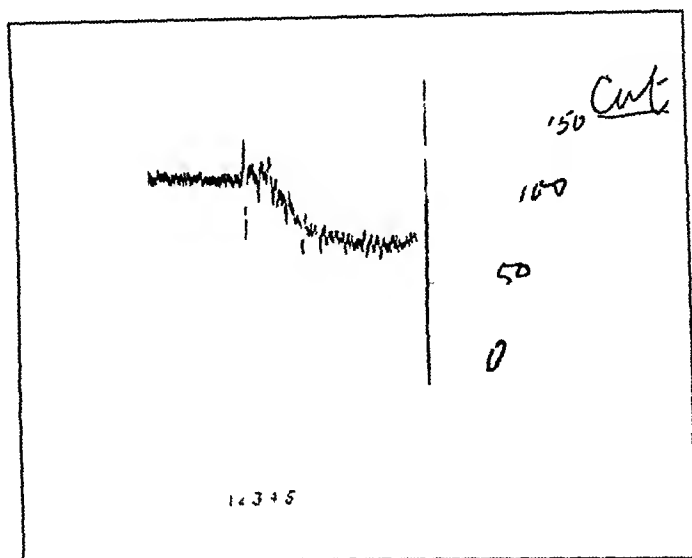


FIG 401—Intramuscular injection of histamine Cat 87 2500 grm Chloralose Tracheotomy Initial blood-pressure 120 mm Hg Signals 1, 2, 3, 4, 5 10 mgrm histamine intramuscularly right thigh Blood-pressure 75 mm Hg Time signal 30 seconds

which can be obtained from the complete destruction and intensive extraction of more than 8 kilo of muscle The difficulty, therefore, of assigning to the damaged muscle the rôle of producing an amount of toxins adequate to produce a lasting fall of pressure cannot be dismissed too lightly

THE EXPERIMENTS

One hundred cats were used as the subjects of experiments The animals were anesthetized by chloralose (0.08 grm per kilo) given intravenously in 10 c c of water The blood-pressure was recorded by a carotid cannula and a mercury manometer Trauma was inflicted by twenty blows of a heavy iron bar on one thigh The skin remained intact, but in each instance the femur was broken, and at autopsy considerable injury to the muscles of the thigh could be demonstrated No animal was allowed to recover from the anæsthetic, and in all cases a post-mortem examination of the abdominal and thoracic viscera was carried out In estimating the increase in weight of the traumatized extremity, the lower half of the cat was amputated by a circular incision passing through the lower lumbar vertebræ, and the portion so removed was bisected by section of the symphysis pubis and the sacrum

Our object was to reproduce experimentally the conditions found in the fatal cases that we had observed in practice The milder degree of trauma which has

been used by some other experimentalists, achieved by repeated blows so graduated as to crush the muscles of the thigh but not to break the femur, produces a condition of the limb which has no doubt its physiological interest, but a condition which has rarely, if ever, its counterpart in surgical practice and certainly fails to conform to the type case on which this research is based

SECTION I—THE AREA OF TRAUMA AS A SOURCE OF TOXINS

Cannon and Bayliss demonstrated that the fall in blood-pressure which develops within about twenty minutes after local trauma to the thigh in anæsthetized cats fails to develop if, prior to trauma, ligatures are applied to the iliac artery and vein. In the only experiment recorded, the ligatures were removed after thirty-three minutes. The restoration of the blood-flow was attended by an immediate fall of pressure. They explained this phenomenon by the inability of a hypothetical depressor toxin to get into the general systemic circulation before the ligatures were removed. Our own experiments bearing on the supposed liberation of toxic substances are summarized under four headings, as follows

I Occlusion of Venous Return.—Parsons and Phemister found that trauma to an extremity, the femoral vein of which has been ligated, results in more rapid fall in blood-pressure and swelling of the limb than when the venous return is not obstructed. This is adduced as evidence against the theory that shock was caused

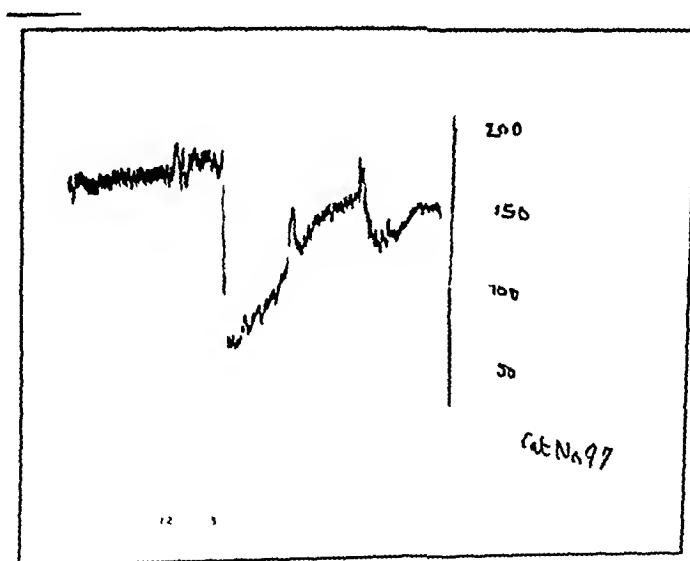


FIG. 402.—Ligation of common iliac vein and injection of histamine. Cat 97 2500 grm Chloralose. Tracheotomy. Initial blood-pressure 170 mm Hg. Signal 1-2 Ligatures on right common iliac vein and section of vessel. Signal 3 1 mgrm histamine injected into tributary of right femoral vein. Blood pressure falls to 75 and recovers. Time signal 30 seconds.

by a circulating toxin. Holt and Macdonald used ligation of the common iliac vein to prevent the passage of blood from the traumatized tissues into the circulation. But the following experiment shows that ligation of either the femoral or the common iliac vein is insufficient to prevent completely the venous return from the limb.

The right common iliac vein was exposed and divided between ligatures. Histamine injected into a tributary of the right femoral vein or into the thigh muscles produced a characteristic fall in blood-pressure (*Fig 402*). Also, air introduced into the femoral vein post mortem passed readily into the vena cava by several anastomotic paths. Effective occlusion of the venous return was however attained by ligature of the femoral vein and its tributaries in the groin and the external, internal, and common iliac veins, and the inferior vena cava and its transverse ilio-lumbar tributary. After this procedure, injection of histamine into the thigh failed to affect the systemic blood-pressure. In a series of experiments, trauma applied to a limb with venous ligatures as above detailed evoked a rapid decline of blood-pressure and the appearance of a state of severe shock with an early fatal termination. In some experiments a tourniquet was applied tightly to one limb, only the femoral artery being excluded. Trauma to the thigh distal to the tourniquet produced a marked degree of shock and early death.

2 Perfusion of the Area of Trauma.—The searching analysis of the action of histamine and the detailed investigation of the production of histamine by Dale and his collaborators provided a basis for the theory that wound shock was caused by absorption of toxic products from damaged muscles. M I Smith has shown that blood removed from the clamped femoral vein of a traumatized limb and re-injected into the animal caused a rise and not a fall of blood-pressure. This observation was controlled by injecting a histamine solution peripherally into the clamped femoral artery and demonstrating a depressor response on injecting blood removed from the femoral vein of that side into the opposite femoral vein. Smith also found that a direct transfusion from the femoral vein of a traumatized limb into a second animal was not associated with any depressor effect on the blood-pressure of the recipient.

We have performed a series of experiments on perfusion of a traumatized limb and failed to elicit any evidence of the presence in the traumatized tissues of vasodilator substances to which might be attributed the general vascular collapse of shock.

Perfusion of the limb was carried out in the following manner. An incision was made over the femoral artery in the groin.

The vessel was dissected and ligatured. A cannula inserted into the peripheral segment of the femoral artery connected with a reservoir from which physiological saline at a constant rate and at body temperature could be supplied. This was secured by having an inverted flask of saline in a box, the temperature of which was kept constant by two filament lamps and a simple thermostatic device so

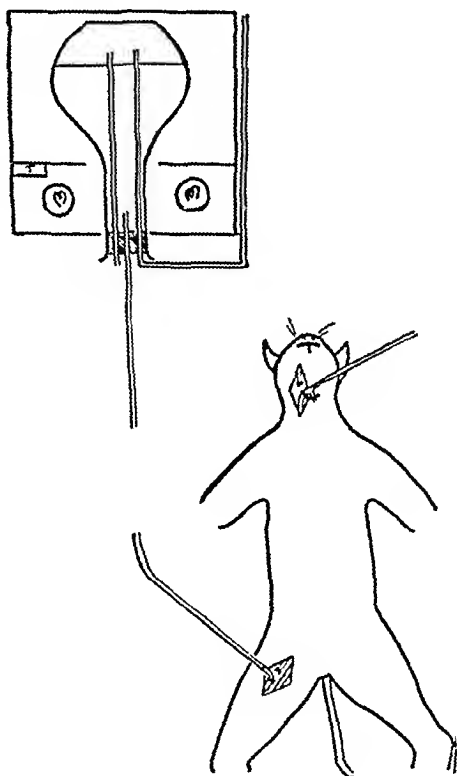


FIG 403.—Perfusion of limb experiment

adjusted that the temperature of the saline at the cannula was kept accurately at body temperature. The arrangement of the apparatus for these experiments is shown in *Fig 403*. The limb was then traumatized and, at definite periods after trauma, saline was allowed to flow into the femoral artery. A rise of blood-pressure was evoked, so that there was no evidence that any depressor principle was being carried into the circulation by the saline perfusing through the traumatized tissues. Saline infusion through the area of trauma was carried out immediately after trauma or at varying later periods, in some experiments a period of four hours elapsed. A possible criticism is that the *pressor* effect of the volume of the saline infusion neutralized the *depressor* effect of any vasodilator substances present. Histamine was therefore introduced into the traumatized area and on repeating the infusion, the rate of which was kept constant throughout the experiment, an immediate fall of pressure was produced (*Fig 404*).

As a further control a solution of histamine equivalent to 2 mgrm of the base was introduced into the thigh muscles of a cat after ligature of the external iliac artery. Thirty minutes later a cannula was inserted into the artery distal to the ligature and saline infused into the animal. A typical histamine response was produced, an immediate sharp drop of pressure with a slow recovery (*Fig 405*). In *Fig 406* is illustrated the production of histamine shock by perfusion after 48 mgrm of histamine had been injected into the traumatized thigh. Perfusion of the traumatized thigh for the same period of time previous to the introduction of the histamine had resulted in a rise in blood-pressure. The site of production of the hypothetical depressor toxins postulated in the toxæmic theory of shock is located in the muscles. Hence it is probably a more strictly accurate reproduction of the actual conditions to introduce the histamine solution into the muscles themselves as we have done in some of these experiments than to inject the drug into the clamped artery as practised by M. I. Smith and by Holt and Macdonald. In one experiment the external iliac artery was tied and the vein clamped. The leg was then traumatized and histamine injected into the area of trauma. When the clamp was removed from the vein and saline was perfused through the femoral artery, the resulting drop of pressure was no greater than that which resulted when an equivalent dose of histamine was injected into an untraumatized thigh and perfusion carried out under exactly the same conditions.

3 The Search for Diffusible Products in the Systemic Circulation — Investigations undertaken to demonstrate depressor substances in the blood of shocked animals have been uniformly unsuccessful. Schneider prepared extracts of the blood of shocked patients and tested these for depressor activity with negative results. Normal serum he found contained a principle which induced contractions of the isolated intestinal muscle preparation of the guinea-pig. This principle, which has no depressor quality, is increased in cases of trauma and extensive burns. Small quantities of histamine added to the blood were recoverable by his method of extraction. Hence the blood of shocked patients contained no histamine-like substance. Other investigators failed to find any evidence of a toxin on injecting blood of shocked animals into normal animals. It was pointed out in the introduction that the experiments of McIver and Haggard are susceptible of a different interpretation from that which they postulated. The fall of pressure in an animal, the femoral vessels of which are utilized to supply blood to the amputated lower

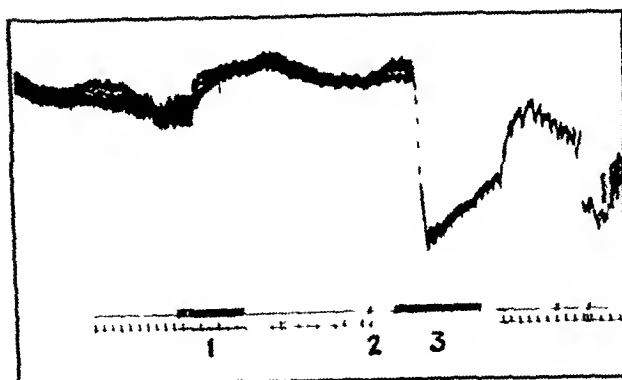


FIG 404—Cat 2 Saline perfusion of traumatized limb Trauma to thigh—vessels clipped
Signal 1 Perfusion of traumatized limb 30 minutes later Signal 2 2 mgrm histamine into traumatized
area Signal 3 Perfusion of traumatized limb Time signal 30 seconds

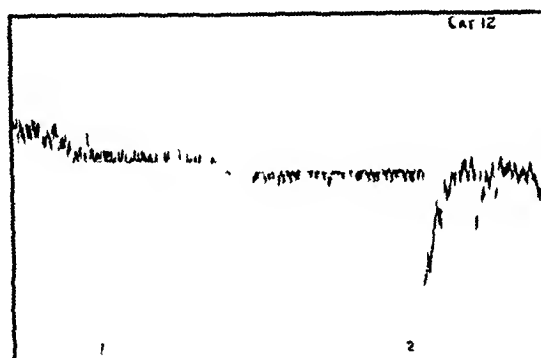


FIG 405—Perfusion of limb thirty minutes after injection of histamine Cat 12 3250 grm
Chloralose Tracheotomy Signal 1 External iliac vessels clipped Two mgrm of histamine into
thigh muscles (L) Signal 2 Perfusion of limb (L) Time signal 30 seconds

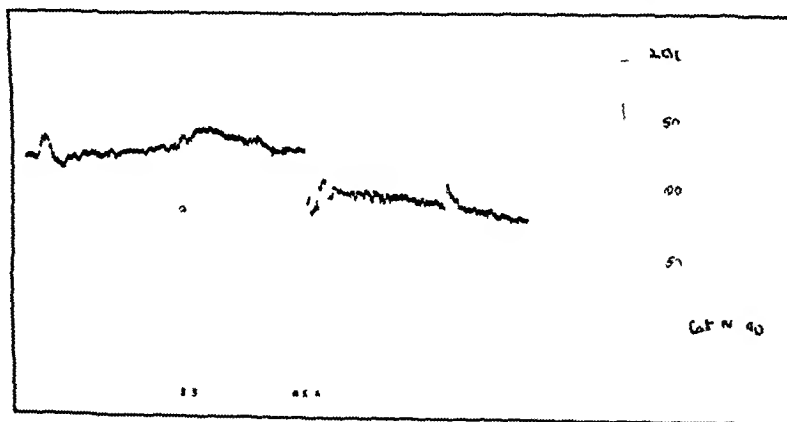


FIG 406—Perfusion of traumatized limb—injection of 4750 grm Ligation of aorta, ilio-lumbar, middle sacral and c and its branches in the groin Clip on external iliac vein Sig external iliac vein Signals 2-3 Perfusion with 40 c c saline histamine into traumatized area Signals 5-6 Perfusion with 40 c c saline for 160 seconds Time signal 30 seconds

Cat 90
al artery
clips off

48 mgrm

extremity of a second cat, on trauma to that extremity is as readily attributable to hæmorrhage into the traumatized tissues as to the absorption of a toxin

Cross-circulation experiments are open to the criticism that the entire preparation tends to act as one unit. Fall of blood-pressure in one animal, whatever be its cause, results in bleeding from the second animal into the first until the pressures reach an approximate equilibrium. To obviate this difficulty we adopted a method based on vividialysis. The apparatus designed for this purpose is shown diagrammatically in Fig 407. It consists essentially of two glass tubes (A and B), 15 cm long, of 10 mm cross-section, fitted at each end with rubber stoppers. The two limbs are connected together at one end by a short transverse tube, and each is furnished close to the opposite end with a short tube at right angles (D and E). Contained within each limb of this outer circuit is a tube of collodion membrane 8 mm in diameter (P and Q), each securely ligatured at its end to the short glass tubes (R, S, T, and U) passing through the rubber stoppers. T and U are connected together by a short piece of rubber tubing. R and S serve as inlet and outlet for the inner circuit. There are thus two distinct U-shaped circuits within the apparatus. Cannulae in the vessels of one animal are connected to R and S and of the other animal to D and E. The circuit of the blood of the one animal is distinct from that of the other except in so far as diffusion occurs across the collodion membranes. If one animal is traumatized, the presence of any depressor toxic product in its general circulation should presumably be recognized by a fall of blood-pressure in the second animal. The toxin would pass across the membrane from the circulation of the shocked animal to that of the other.

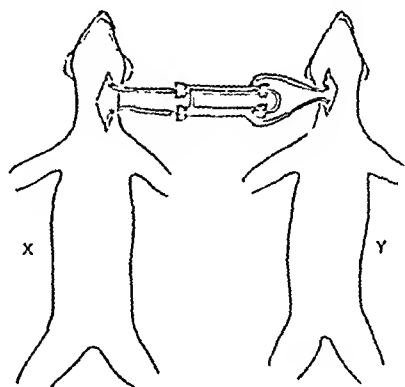
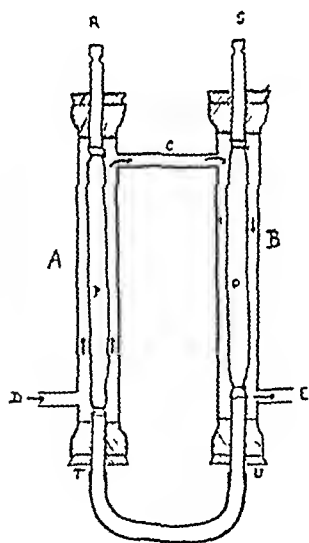


FIG 407—Vividialysis

For each experiment with this apparatus two dogs were used. Novirudin was administered as an anticoagulant. The carotid artery on one side was exposed and dissected in the neck of animal X. Clamps were applied to its upper and lower ends. The artery was then divided and two cannulae were inserted, one directed proximally, the other distally. The inner circuit of the apparatus (within the collodion membranes) was filled with physiological saline and the cannulae connected by paraffined tubing to the inlet and outlet tubes (R and S in Fig 407). The clamps were then removed. The integrity of the membranes and the free

flow of blood within them was verified. In a similar manner the second animal (Y) was connected to the outer circuit (limbs D and E). The fact that the blood was flowing freely could be established at any stage of the experiment by observing the pulsations in the outlet limbs or by feeling the pulse in the peripheral segment of the carotid. The blood-pressure of each animal was recorded from the femoral artery.

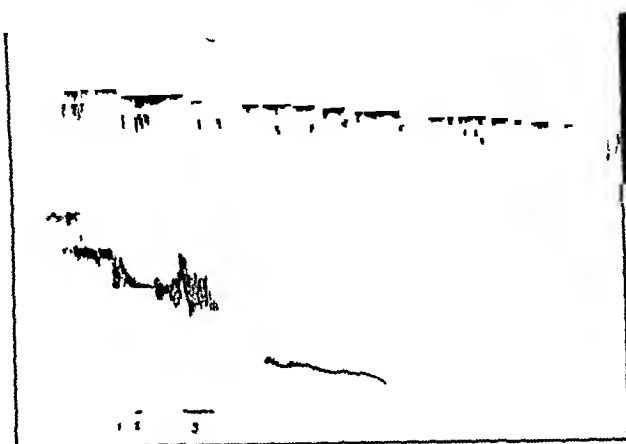


FIG 403—Dogs 1, 2 Vividialysis Trauma. Each dog weighed 1500 gm and the anæsthetic was morphia and nembutal. The two animals were connected to the dialyser and the circuits opened at Signals 1 and 2. Dog 1, lower curve; Dog 2, upper curve. Signal 3 Trauma to Dog 1. The traumatized dog went into shock and died thirty minutes later, while the blood-pressure of Dog 2 remained unaffected. Time signal 30 seconds.

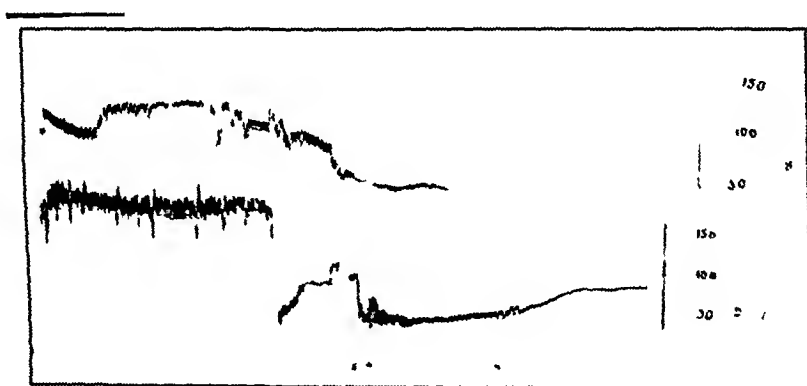
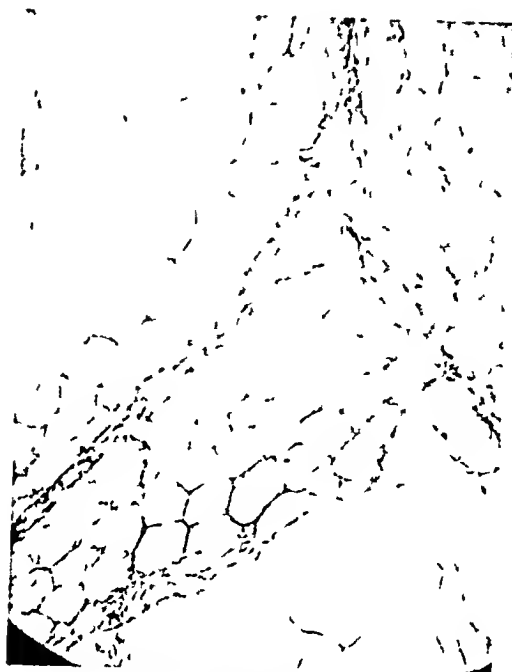


FIG 409—Dogs 7, 8—Vividialysis Histamine poisoning. Two dogs anesthetized by morphia and nembutal connected to the dialyser and at Signal 1 circuit opened. Dog 7, lower curve; Dog 8, upper curve. Initial blood pressure Dog 7, 170; Dog 8, 130. Signals 2-7 Successive doses of 5 mgrm histamine in 10 c.c. water injected into the femoral vein of Dog 7. An immediate fall in blood-pressure which was maintained at a level of 40-60 for about an hour. The blood pressure of Dog 8 declined, and 15 minutes after the injection of histamine into Dog 7 it was at 60, and 45 minutes later Dog 8 died. Both showed at autopsy the evidences of histamine poisoning.

Using this apparatus we were able to observe in a series of experiments (a) That trauma to Dog Y is without effect on the pressure of Dog X, and (b) That slow continuous intravenous infusion of histamine maintaining the pressure of Dog Y at shock level is accompanied by a fall of pressure in Dog X (Figs 408, 409). These experiments appear to weigh strongly against the conception of any humoral



A (x 80)



B (x 80)



B (x 190)

FIG 410—Microphotographs of omentum in A, Traumatic shock, B, Histamine poisoning

agency, either produced locally in the area of trauma or more remotely, bearing a causal relationship to the development of traumatic shock. One reservation must be mentioned. The circulation toxin might be of so complex a molecular structure as to be incapable of dialysis across a collodion membrane. Only one active vasodilator principle identified in tissue extracts is known to be non-dialysable. The chemical nature of kallikrein, as it is called, is not yet known. It has been identified by characteristics of physiological activity different from those of histamine. Significantly for our purpose, it is rapidly inactivated by normal serum.

4 Pathological Picture in Histamine Shock and Traumatic Shock —

The post-mortem appearances in animals whose death has followed the administration of histamine differs significantly from those seen after death from traumatic shock. After histamine poisoning general examination of the abdominal viscera discloses striking features. There is a dusky diffuse congestion of the intestines. The cut surfaces of the liver and kidneys bleed readily. The omentum is engorged and presents large vessels macroscopically visible in the fat streaks. There is an intense oedema of the pancreas. The spleen is generally blue in colour and small in size. The lungs are congested with dark-coloured blood. The heart is well filled.

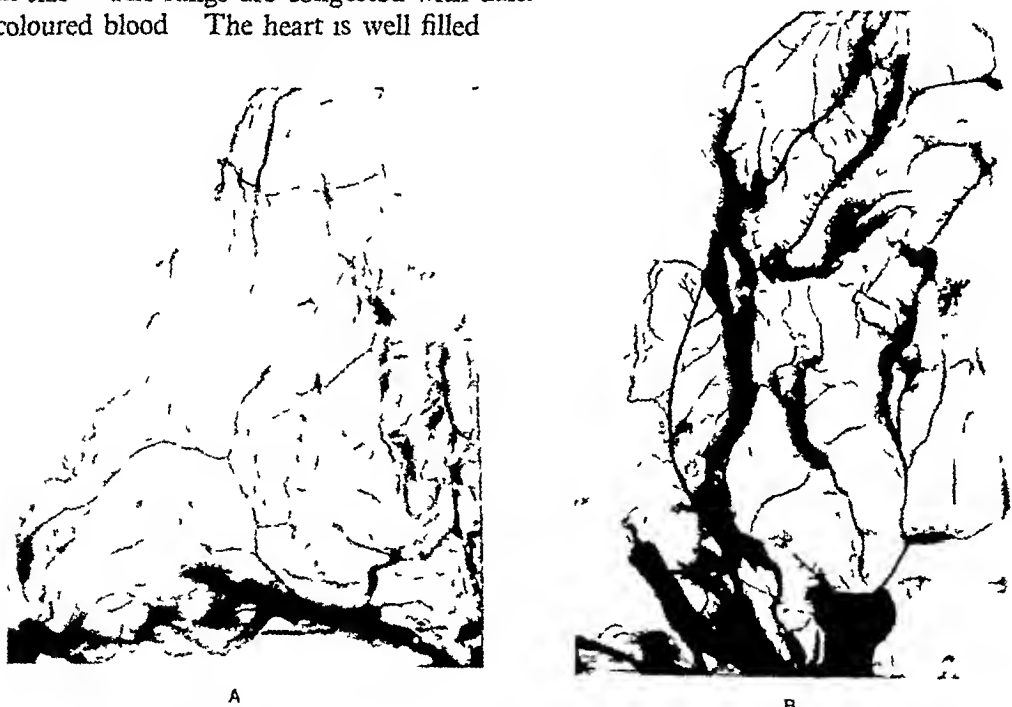


FIG 411 —Macroscopic specimens of omentum in A, Traumatic shock, B, Histamine poisoning

In striking contrast to this, the characteristic feature after death from traumatic shock is a general pallor of the viscera, especially of the intestines, and of the omentum. The liver and kidneys do not bleed readily when cut. Oedema of the pancreas is never observed. The spleen is contracted, but red in colour. The lungs are generally pale. The heart contains little blood. Frequently the left ventricle is in a state of tonic contraction.

Rich examined the capillaries in the omentum in cats. He demonstrated that when histamine is injected in amounts sufficient to produce shock a progressive

dilatation of the visible capillaries is observed, with opening up of a large number of occult capillaries. There is also a dilator effect on the immediately adjacent arterioles and venules. This constitutes a direct confirmation of the site of the action of the drug, as postulated by Dale. We have examined preparations of the omentum fixed *in situ* by flooding the peritoneal cavity with formalin-saline in animals subjected to trauma and in others after toxic doses of histamine. The dilatation of the peripheral vascular bed in histamine shock offers a striking contrast to the extreme degree of contraction of the capillaries and minute vessels in the case of traumatic shock. On microscopic examination under high power the dilated capillaries can only be found with difficulty after careful search in the 'shock' omenta, even the larger vessels are often very poorly filled with blood, as shown in the microphotographs (Fig 410). The macroscopic contrast is well seen in cleared specimens of the omentum (Fig 411).

SECTION II—FLUID LOSS INTO THE AREA OF TRAUMA

In the experiments that provided the main basis for the toxic theory of shock, Cannon and Bayliss found that the fluid loss into the lacerated tissues was inadequate to account for the fall in blood-pressure produced by traumatizing one of the hind limbs of anæsthetized cats. As already mentioned, Blalock and Parsons and Phemister repeated these experiments and showed that the local fluid loss extended beyond the area of trauma into the groin and flank. They replaced the Cannon and Bayliss method of amputating by symmetrical incision through the upper thigh by amputation well above the area of swelling. The local fluid loss due to extravasation of blood and exudation of plasma, as expressed by the difference in weight of the injured and non-injured limbs after this hindquarter amputation, was sufficiently great to permit the conclusion that fluid loss itself sufficed to account for the decline in blood-pressure. Blalock found that he could produce the phenomena of shock in his animals by removing a corresponding amount of blood from an artery even when the animals suffered no trauma.

In Table I the details of a series of eight traumatized animals are given. Fig 412 is a kymograph record of the course run in one of these animals. The average fluid loss into the traumatized limb in these animals, expressed as a percentage of the calculated blood volume, is 36 per cent. Macdonald and Holt give 51 per cent.

Table I—CONTROL EXPERIMENTS

CAT NO	WEIGHT OF CAT	INCREASE IN WEIGHT OF TRAUMATIZED LIMB	FLUID LOSS EXPRRESSED AS A PERCENTAGE OF CALCULATED BLOOD VOLUME	INITIAL BLOOD-PRESSURE	B P AFTER ONE HOUR	SURVIVAL TIME
	Grm	Grm		mm Hg	mm Hg	Hours
69	1500	55	48 per cent	160	60	4½
78	2500	58	30 per cent	195	150	3
88	2750	—	—	180	140	3½
83	3000	130	56 per cent	160	20	1½
50	3200	56	22 per cent	170	50	2½
76	4250	—	—	165	150	6½
20	3000	69	30 per cent	165	90	2
92	3000	53	22 per cent	135	110	2

for dogs and 43 per cent for cats. Including several other experiments not shown in the table, we obtained an average figure for the fluid loss in 12 cats of 32 per cent. When very severe trauma has been induced the fluid loss is sufficiently great to dismiss the need to invoke any other agency contributing to the production of shock. In some of our experiments, however, the fluid loss amounted only to about 20 per cent of the total blood-volume. In spite of this, the onset of shock in these animals was comparable in every way to that obtained by Blalock, both in degree of severity and in the rapidity with which death occurred. The degree of fluid loss into the traumatized tissues does not bear a direct relation to the survival time. These findings suggest that some other factor might be operative.

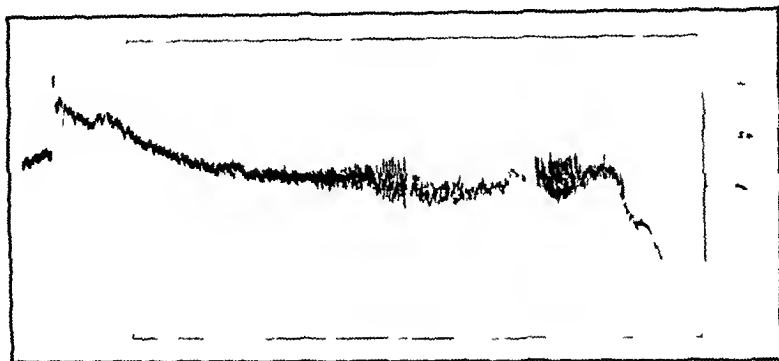


FIG 412—Trauma to thigh. Cat 92. 3000 gm. Chloralose. Tracheotomy. Initial blood-pressure 135. Signal. Trauma to thigh. Death 2 hours 20 minutes after trauma. 53 gm increase in weight of traumatized limb. Time signal 30 seconds.

Many workers have shown that occlusion of the main vessels of a limb prevents the development of traumatic shock. We have found that when such occlusion is effected merely by ligature of the external iliac vessels a gradual decline in pressure manifests itself if the experiments are followed for an adequate period. Examination of the lacerated tissues and a comparison of the weights of the traumatized limb and of its fellow demonstrate that this procedure does not completely prevent hæmorrhage into the injured area. Collateral channels are obviously still operative. Complete occlusion of the blood-supply to a limb is only obtained by ligature of the abdominal aorta, the ilio-lumbar artery, the middle sacral, the external iliac, the profunda femoris, and the femoral artery and its branches in the groin. Complete obstruction of the venous return is only achieved by ligature of the inferior vena cava and the veins which correspond to the arteries enumerated above. To avoid repetition, we refer in the course of this account to such a preparation as an 'anæmic limb'. The total separation of such a limb from the general circulation can be demonstrated. Drugs injected into the muscles of such an anæmic limb are not absorbed into the general circulation. Trauma to the anæmic limb had no marked effect on the blood-pressure, although the animals were followed for many hours. An experiment in this group is illustrated by Fig 413. The initial blood-pressure in this cat was 190 mm of Hg, and seven hours after trauma was still 180 mm of Hg. In the experiments in this group the animals were

followed for periods of eight to twenty hours, and in no case had the blood-pressure declined to a shock level

The toxic factor having been excluded, these results have been used as evidence for attributing shock purely to fluid loss, since it is argued that shock fails to develop despite the fact that the nervous paths are intact. The integrity of normal pathways for nervous impulses from such a limb has been tacitly assumed. We find that after complete occlusion of the blood-supply the knee-jerks can no longer be elicited after half an hour, although the muscles still respond to direct stimulation. In short, the question to be settled is whether the 'anæmic' limb is not in fact also 'anæsthetic'. Although a much more detailed investigation is necessary before a final conclusion can be reached as to the functional efficiency of nerve endings and nerve trunks in the presence of an intense anæmia, it is unwise to assume that they are functioning normally. Although the sudden onset of shock on removal

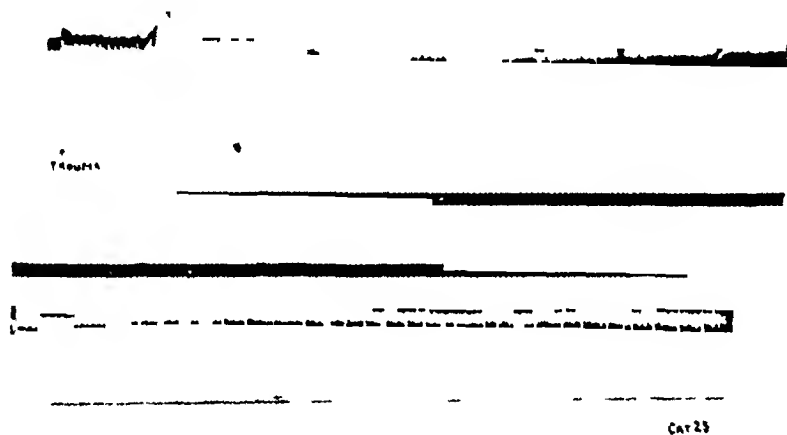


FIG 413—Trauma to thigh after ligation of vessels. Cat 25 3000 grm Chloralose Tracheotomy Initial blood pressure 190 Signal Trauma to thigh Killed 5 hours later with a blood pressure of 180 Difference in weight of limbs, 17 grm Time signal 30 seconds

of a tourniquet from a wounded limb might be attributed solely to the sudden loss of fluid as advocated by Blalock, it is also feasible that the sudden restoration of a blood-supply might be the signal for releasing a flood of nervous impulses.

Experimental support of this concept is provided by the following experiment. A cat (No 43) was prepared in the usual way, and his right hind limb was rendered anæmic. A second cat (No 42) was prepared. An anastomosis of the central end of his femoral artery to the peripheral end of the divided femoral artery of Cat 43 and a venous anastomosis were made simultaneously. In this way the anæmic limb of Cat 43 received a new blood-supply from the donor cat (42). The limb was then traumatized, as in other experiments. Trauma was followed by a progressive decline of the blood-pressure of Cat 42. His death followed some two hours later. Soon after this Cat 43 went into shock and died. At autopsy the organs of Cat 43 dripped blood on section. It was thus clear that fluid loss had not contributed to death. The efficiency of the vascular occlusion had previously been established by the injection of histamine into the anæmic

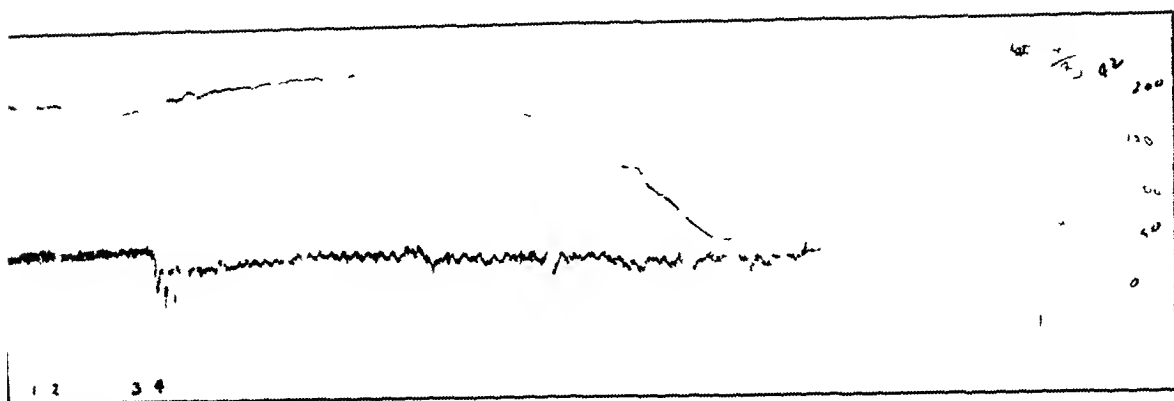


FIG 414—Cats 42, 43 Trauma to a limb excluded from the circulation but supplied with blood from a second animal Cat 42 (upper tracing) 3500 grm Chloralose Tracheotomy Cat 43 (lower tracing) 3250 grm Chloralose Tracheotomy Signal 1, 2 1 mgrm histamine into thigh of Cat 43 Signal 3 Vascular anastomosis opened Signal 4 Trauma to Cat 43 Signal 5 Massage of traumatized limb (Cat 43) Time signal 30 seconds

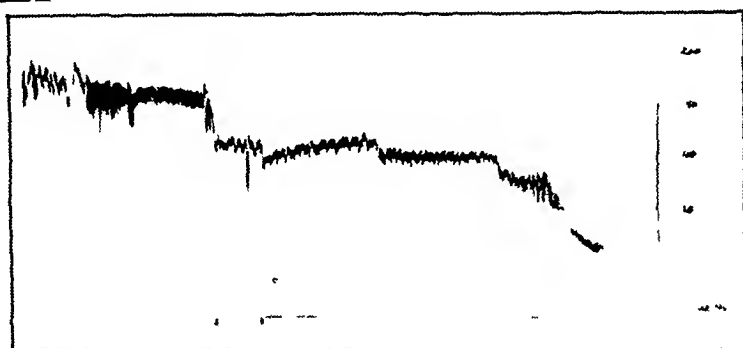


FIG 415—Ligature of vessels to limb—trauma—hemorrhage Cat 93 3500 grm Chloralose Tracheotomy Initial blood-pressure 185 mm of Hg Vessels to limb tied Signal 1 Trauma After 30 minutes blood pressure still normal level Signals 2-4 10 c c of blood from subclavian artery Signals 5-7 15 c c of blood from subclavian artery Death after loss of 60 c c of blood Time signal 30 seconds

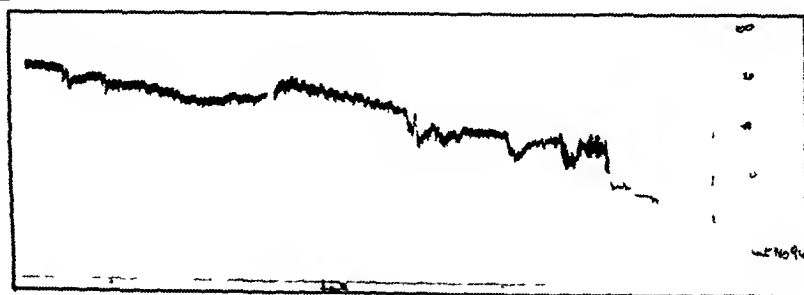


FIG 416—Effect of hemorrhage on normal cat Cat 94 3500 grm Chloralose Tracheotomy Initial blood pressure 165 mm of Hg Signal 1, 2 30 c c of blood from subclavian artery Signal 3-12 15 c c of blood from subclavian artery Death after the loss of 105 c c of blood Time signal 30 seconds

limb As there was no evidence of any incidental cause of death we are left with the presumption that the only remaining factor—nervous impulses from the traumatized area—led to the fatal event (*Fig 414*)

A second type of experiment supports this view Although anæmia of the traumatized limb prevents the onset of shock and a decrease in blood-pressure, the animal is affected by the trauma in a way which is not immediately demonstrated on the blood-pressure tracing The limb is rendered anæmic, and at once—while the knee-jerks still remain active—trauma is applied and as usual there is no change in the level of the blood-pressure tracing, indeed, one hour after trauma its level was still, as originally, 180–190 At this point 15 c c of blood was removed from a cannula previously inserted into the subclavian artery There was an immediate fall in blood-pressure and this fall persisted and was increased by successive bleedings, each of 10 c c, until 60 c c had been removed, when the animal died A control cat of equivalent weight not only withstood the loss of 105 c c before death ensued, but after each successive bleeding made a recovery—the difference in response of the two animals is shown in *Figs 415, 416*

SECTION III—THE AREA OF TRAUMA AS A SOURCE OF NERVOUS IMPULSES

It is not our present intention to discuss the vexed question of primary and secondary shock We are merely concerned with the course run by our traumatized animals That course, like the clinical course of the patients whom we observed during this inquiry, did not permit of such a division It has usually been assumed that the initial fall in blood-pressure after trauma is due to a nervous mechanism Under some conditions this may be true, but often the initial fall is due to hæmorrhage into the traumatized area We found that trauma to the anæmic limb was seldom followed by an initial drop in pressure even when this trauma was inflicted at a stage when a reflex arc, as demonstrated by the knee-jerks, was still present On the other hand, in some of the animals subjected to a preliminary section of the cord or given spinal anæsthesia the initial drop was as marked as in the control animals, although the knee-jerks were absent (*Figs 417–419*)

The relation of the nervous system to the syndrome of traumatic shock has been investigated in a series of experiments which fall into the following groups (1) Section of the nerves to the limb, (2) Section of the spinal cord, (3) Section and destruction of the spinal cord, (4) The induction of spinal anæsthesia On examination of the four groups of experiments one fact emerges quite clearly when an attempt had been made to exclude the nervous discharge from the area of trauma, the syndrome was less severe than in the control group In the control group only one cat survived for as long as 6 hours, the average survival period was 3 hours 12 minutes, and each animal showed an appreciable drop in blood-pressure—in some cases 100 mm of Hg—at the end of the first hour after trauma In the group subjected to nerve section most of the animals ran so long a course that it was impracticable to follow them all to death The average survival time of those which were followed to death was 5 hours 54 minutes This includes the group subjected to cord section—an operation which in itself entails additional trauma—and the course run by those animals subjected to preliminary nerve section or spinal anæsthesia was much more favourable We would emphasize in considering

the importance of the nervous factor that it is the course run by the animal over a period of hours rather than his *immediate* response to trauma which is significant. Perhaps some recent workers have failed to appreciate the nervous aspect of the syndrome because they have failed to appreciate this point.

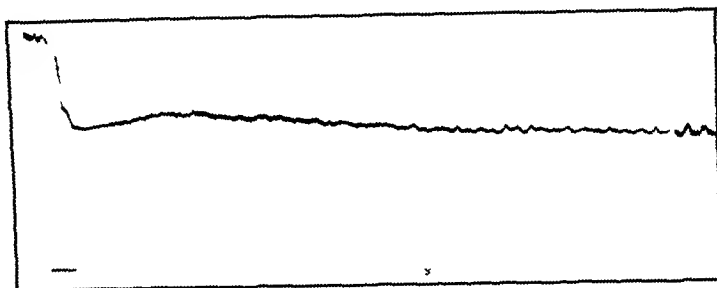


FIG 417—Trauma to thigh marked initial fall in pressure. Cat 50 3250 grm Chloralose Tracheotomy Initial blood-pressure 170 mm of Hg Signal Trauma Blood-pressure falls to 50 Death after 2 hours 20 minutes Increase in weight of traumatized limb 56 grm Time signal 30 seconds

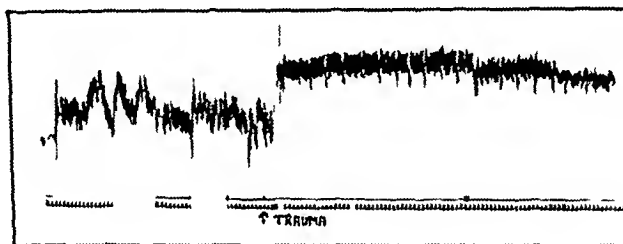


FIG 418—Ligature of vessels—trauma—rise in pressure. Cat 35 3000 grm Chloralose Tracheotomy Ligature of all vessels to thigh Initial blood-pressure 190 mm of Hg Signal Trauma Pressure rises to 200 Time signal 30 seconds

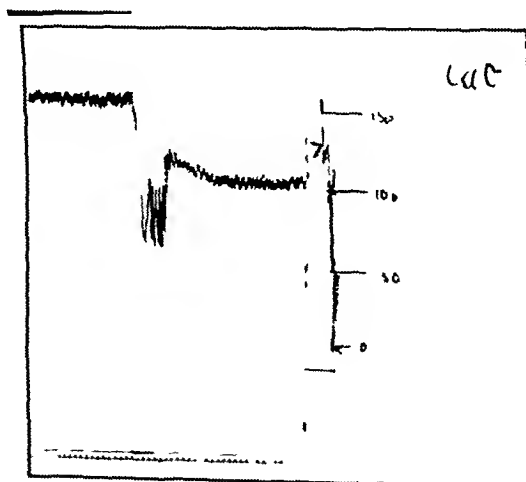
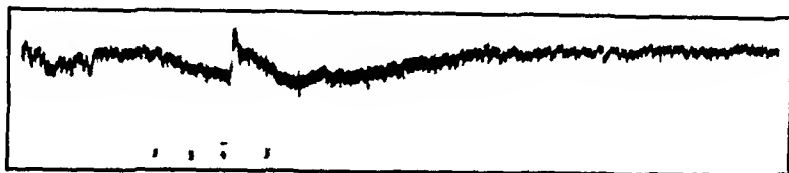


FIG 419—Trauma to thigh after section of spinal cord—marked initial fall in pressure. Cat 62 4000 grm Chloralose Tracheotomy Laminectomy and section of cord at D 12 Blood-pressure 145 mm of Hg Signal Trauma Blood pressure falls to 50 Time signal 30 seconds

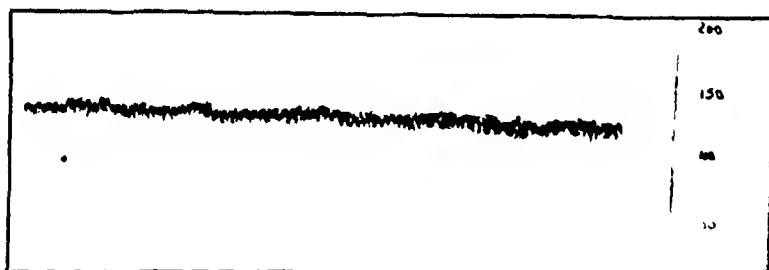
1 Section of the Nerves to the Limb—Simonart's criticism of the original experiment by which Cannon claimed to demonstrate the onset of traumatic shock despite the preliminary section of the femoral and sciatic nerves has been summarized in our introduction. We repeated this experiment with two modifications: the obturator nerve was cut as well as the femoral and sciatic nerves, and as a preliminary to section the nerve trunks were infiltrated with 1 per cent novocain



A



B



C

FIG. 420.—Section of femoral, sciatic, and obturator nerves. Trauma. Cat 99 3000 grm Chloralose 100 mg. Section of femoral, sciatic, and obturator nerves. Blood pressure 135 mm of Hg. A. Signal 1. Clip off external iliac artery. Signal 2. Clip off external iliac artery. Signal 3. Clip on external iliac artery. Signal 4. Clip off external iliac artery. Signal 5. Clip off external iliac artery. B, The record 3 hours after trauma. C 5½ hours after trauma. Blood pressure 125. Animal killed. Increase in weight of traumatized limb 40 grm. Time signal 30 seconds.

When these conditions were fulfilled shock *failed* to develop. One cat was killed 6½ hours after his trauma with a blood-pressure of 130 (Initial BP 110) and another was killed 5 hours 20 minutes after trauma with a blood-pressure of 125 (Initial BP 135) (Fig 420). In these two experiments the amount of local fluid loss was admittedly lower than usual (20 per cent), but we have the records of two control cats with an equivalent local loss of fluid, each of which died some two hours after the infliction of trauma. We have already explained our reasons for refusing to accept an exact parallelism between the local fluid loss and the degree of trauma

2 Section of the Spinal Cord.—On the whole the animals subjected to section of the cord as a preliminary to trauma ran a less favourable course than did those subjected to nerve section or spinal anaesthesia. How far this may be due to the trauma involved, and how far to the fact that despite this procedure an effective interruption of all reflex arcs between the thigh and the splanchnic system is not attained, we are not yet in a position to judge. Nevertheless, one of the animals was still at a level of 100 mm of Hg (Initial Blood-pressure 210) when it was killed $7\frac{1}{2}$ hours after trauma, and in this case the fluid loss was 17 per cent of the calculated blood-volume.

3 Section of the Spinal Cord with Destruction of the Lower Segment of the Cord.—In only one of these animals was life prolonged for any appreciable time—it was killed $7\frac{1}{2}$ hours after trauma with a blood-pressure of 85. The local fluid loss in these animals was less than in the control series.

4 Induction of Spinal Anaesthesia.—It became apparent that, in order to test the influence of the nervous system, some method less traumatic than section

Table II—SPINAL ANÆSTHESIA FOLLOWED BY TRAUMA

CAT NO	WEIGHT OF CAT	INCREASE IN WEIGHT OF TRAUMATIZED LIMB	FLUID LOSS EXPRESSED AS A PERCENTAGE OF CALCULATED BLOOD VOLUME	INITIAL BLOOD-PRESSURE	RESULT
39	Grm 2000	Grm —	—	mm Hg 170	Killed after 4 hrs B P 145
18	2250	35	20 per cent	175	Killed after 3 hrs B P 120
17	3000	123	53 per cent	160	B P after 9 hrs, 125 Died after 10 hrs
49	3000	69	25 per cent	120	Died after $8\frac{1}{2}$ hrs (Overdose of anæsthetic)
53	3800	—	—	165	Killed after 4 hrs B P 120
54	4000	89	29 per cent	180	B P after 4 hrs, 100 Died after 9 hrs
61	4000	55	16 per cent	170	B P after 3 hrs, 130 Died after 4 hrs (Overdose of anæsthetic)
16	4500	—	—	140	Killed after $6\frac{1}{2}$ hrs B P 170
46	3250	—	—	170	Killed after 6 hrs B P 140

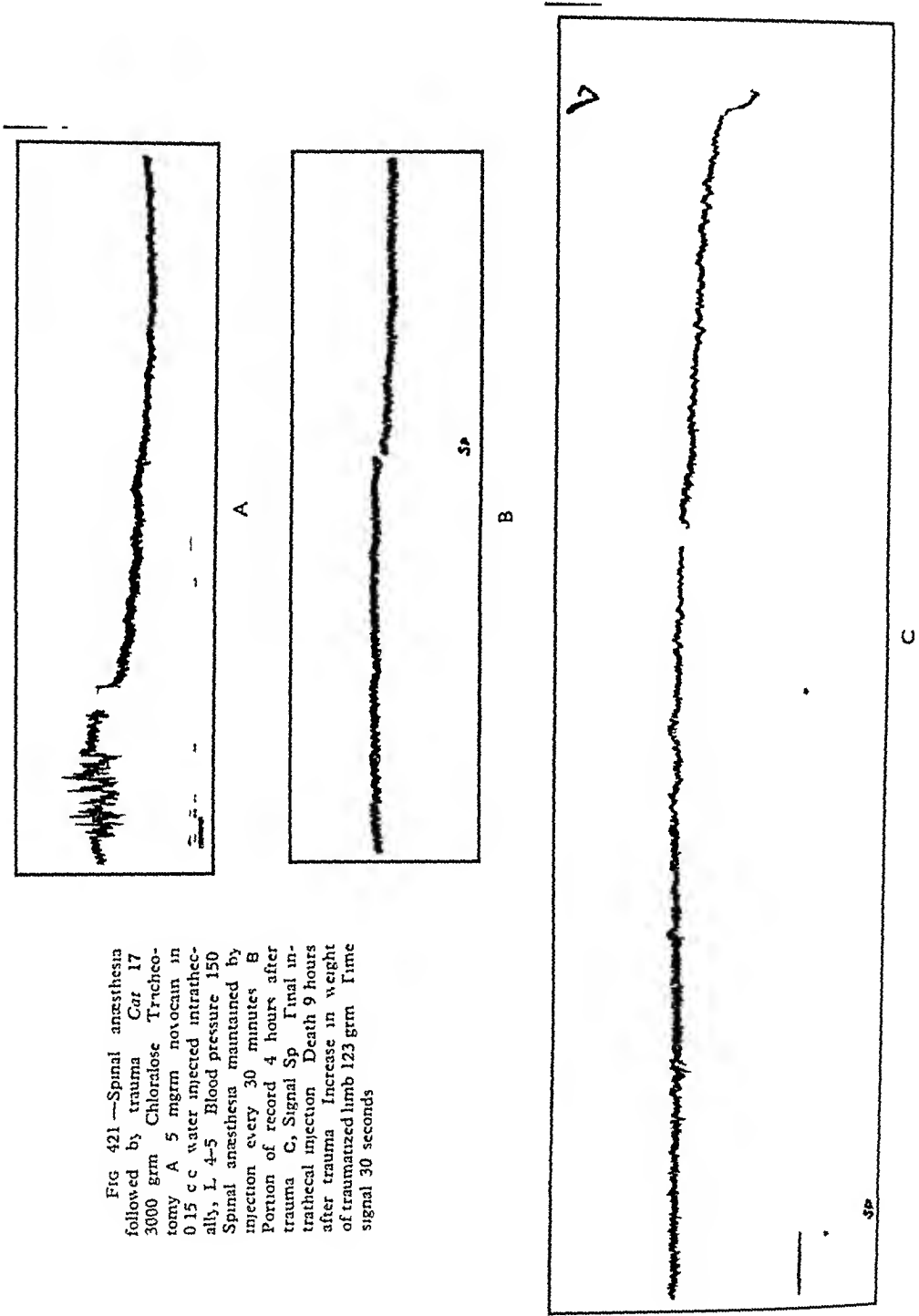


FIG 421.—Spinal anaesthesia followed by trauma Cat 17 3000 grm Chloralose Tracheotomy A 5 mgrm novocain in 0.15 c.c water injected intrathecally, L 4-5 Blood pressure 150 Spinal anaesthesia maintained by injection every 30 minutes B Portion of record 4 hours after trauma C, Signal Sp Final intrathecal injection Death 9 hours after trauma Increase in weight of traumatized limb 123 grm Time signal 30 seconds

or destruction of the spinal cord had to be devised. We also required a method which would effectively exclude the reflex paths between the thigh and the splanchnic region, as the operation of such a local reflex may well be one of the factors responsible for the appearances of the viscera and blood-vessels constantly found at autopsy and on laparotomy of the shocked animal. The only method that seemed to fulfil these conditions was the induction of spinal anæsthesia, and this method was therefore adopted. There is no doubt that this procedure has a most favourable influence in delaying and even preventing the onset of shock. At the same time, although spinal anæsthesia interrupts the nervous paths, both somatic and sympathetic, it has undoubtedly other effects on the vascular system—an increase in blood-volume has been observed by Schneider—and it must be admitted that its beneficial action may be due in part to these effects. For convenience, however, the results of our experiments with spinal anæsthesia in preventing the onset of traumatic shock and influencing its course will be described in this section.

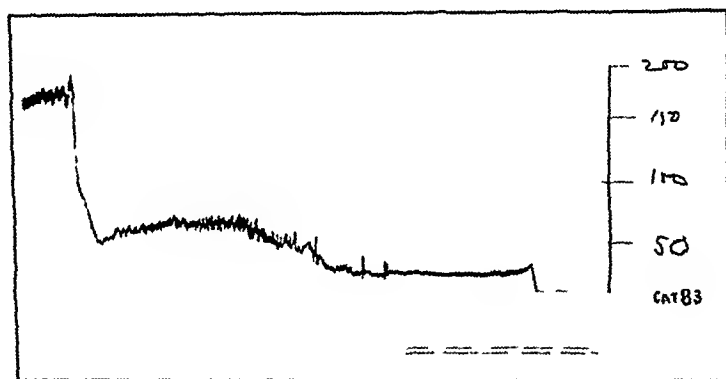


FIG 422.—Trauma to thigh. Cat 83. 3000 grm. Chloralose. Tracheotomy. Initial blood-pressure 160. Signal Trauma. Dead 1 hour 10 minutes later. Increase in weight of traumatized limb 130 grm. Time signal 30 seconds.

In 9 cats spinal anæsthesia was first induced and trauma was not inflicted until the knee-jerk had been abolished. The details of the course run by these animals is shown in *Table II*. In almost every case the blood-pressure at the end of the first hour had declined to a comparatively small extent, whereas in the control group a decline to a shock level (60 mm of Hg) is usual within this period. Only three of these animals died within four hours, and in at least two of these death was probably accelerated by an overdose of intrathecal novocain. The remainder either survived for a period of eight or nine hours and, on cessation of the anæsthesia, went into shock and died, or were killed some six hours after trauma with the blood-pressure at a high level. The blood-pressure of one was higher than at the beginning of the experiment. The course run by *Cat 17* is of particular interest, he survived for 9 hours, despite an unusually high fluid loss into the traumatized area (53 per cent). One of the control cats with a similar fluid loss died in $1\frac{1}{4}$ hours (*Figs 421, 422*). A composite graph comparing the average course run by control animals with that of 'anæmic limb' and spinal anæsthetic animals is shown in *Fig 423*.

Apart from the general decline in blood-pressure there is another characteristic which we have frequently observed. This consists of small variations in pressure shown by small irregularities on the tracing. It is absent from the

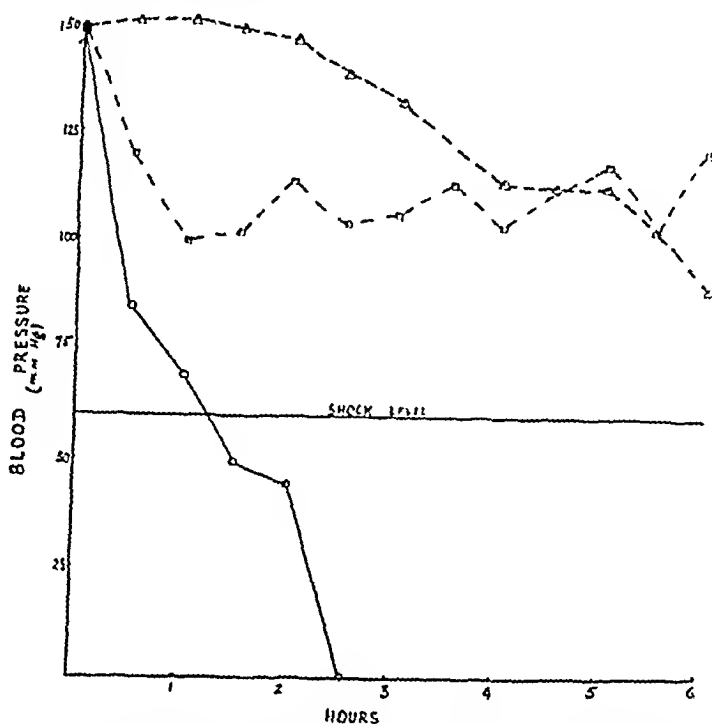


FIG 423—Composite graph of the course of animals subjected to trauma of the thigh. Control= Trauma preceded by spinal anesthesia — — — — — Trauma preceded by ligature of the vessels of the thigh — — — — —

tracing when the traumatized animal is under the influence of an effective spinal anæsthetic. The recovery from the spinal anæsthetic is marked not only by

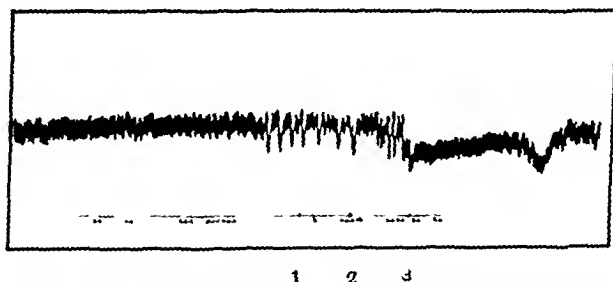
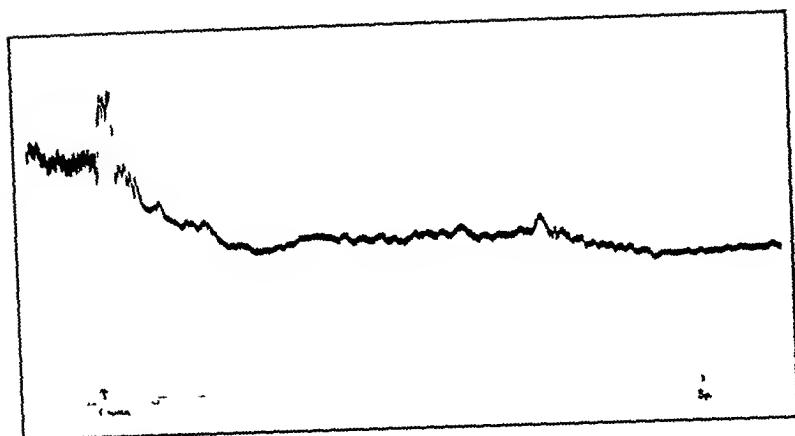
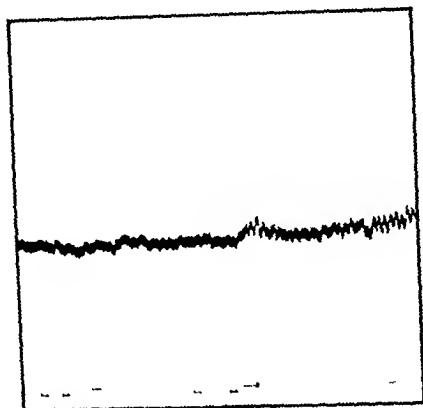


FIG 424—Trauma to thigh. Spinal anesthesia. To show changes in blood pressure. Cat 46. 3750 grm Chloralose Tracheotomy. At Signal 1. Knee-jerks present. Signal 2. Spinal anesthesia. Signal 3. Knee jerks gone. Time signal 30 seconds.

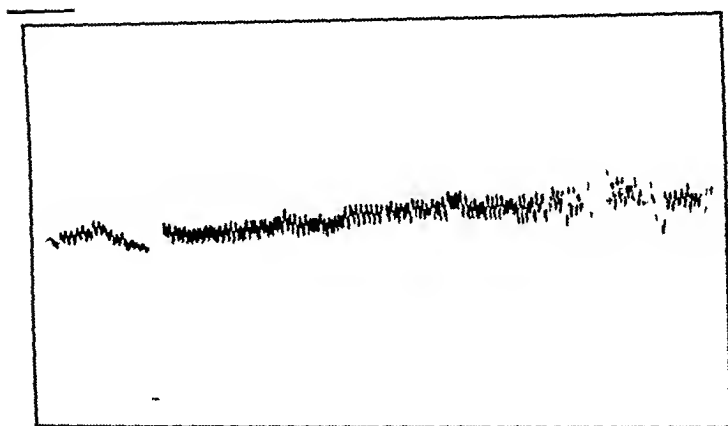
the return of the knee-jerks but by the re-appearance of these small pressure variations. We are not able to state the significance of these variations, but regard them as possible evidence of an abnormal reaction of a vasomotor centre subjected to a barrage of nociceptive impulses from the traumatized area—when



A



B



C

FIG 425 —Trauma followed by spinal anaesthesia. Cat 57. 4000 grm. Chloralose. Tracheotomy. Initial blood pressure 115 mm Hg. A, Signal. Trauma. Blood-pressure falls to 50. Signal Sp. Induction of spinal anaesthesia and this repeated at 30-minute intervals for 3 hours. B, Record 21 hours after trauma. Signal. Induction of spinal anaesthesia. C, Signal. Knee jerks present. Cat killed 5 hours after trauma with a blood pressure of 100. Increase in weight of traumatized limb 116 grm. Time signal 30 seconds.

the barrage is lifted they disappear, only to return as it again becomes effective (*Fig 424*)

Four animals were subjected to trauma and kept until the blood-pressure had assumed a shock level, when spinal anaesthesia was induced (*Table III*). In these cases there was a restoration of blood-pressure to well above a shock

Table III—TRAUMA FOLLOWED BY SPINAL ANAESTHESIA

CAT No	WEIGHT OF CAT	INCREASE IN WEIGHT OF TRAUMATIZED LIMB	FLUID LOSS EXHIBITED AS A PERCENTAGE OF CALCULATED BLOOD VOLUME	INITIAL BLOOD-PRESSURE	B.P. ON INDUCTION OF ANAESTHESIA	RESULT
19	Grm 4750	Grm —	—	mm Hg 200	mm Hg 90	Killed after 8½ hrs B.P. 90
31	4000	131	43 per cent	135	50	Died after 5 hrs
41	3750	90	31 per cent	170	85	Killed after 8½ hrs B.P. 50
57	—	116	—	115	50	Killed after 5 hrs B.P. 100

level, or a prolongation of the survival period, and one of the animals was killed 8 hours after trauma with a blood-pressure of 90 mm of Hg. These results indicate the importance of the nervous factor, and it is of interest to note that the weight differences of the limbs in this group were high (*Fig 425*)

CONCLUSIONS

Again we would emphasize that the conclusions we are about to state apply solely to traumatic shock of the type defined at the beginning of this paper. We are not concerned with the etiology of the shock and collapse which may attend severe burns or trauma to the intestines. Indeed, we feel that a clear conception of the syndrome has been rendered difficult by attempts to establish a common etiological agent for such widely different states as the collapse which may follow a crushing injury of the thigh and the collapse which may be observed in an established case of intestinal obstruction. However, the experimental methods described above, especially the method of vivisection, are peculiarly suitable for the investigation of any syndrome attributed to the circulation in the blood-stream of a toxic product.

Our first conclusion is that a toxæmia due to the elaboration of histamine, or any other depressor substance manufactured in the traumatized area, plays no part in the syndrome of traumatic shock*. We regard the two remaining

* The rejection of the toxic theory must suggest a reconsideration of gas and oxygen anaesthesia as contrasted with anaesthesia induced by ether. It is undoubtedly the fact that etherization leads to a more rapid death in histamine poisoning. This was one of the important considerations that led to the use of gas and oxygen for the shocked patient. The main essential in an anaesthetic, however, is that it should block nociceptive impulses. Unless it can be demonstrated that gas and oxygen forms an equally efficient barrier, its relation to histamine poisoning should no longer be taken into account.

factors, local fluid loss and the discharge of nociceptive nervous stimuli, as the effective etiological agents. The evidence does not allow us to dogmatize as to the relative importance of these factors, although we are inclined to believe that the nervous factor dominates the picture. It is extremely difficult to devise experiments which isolate the action of these two factors. The reaction of the anæmic limb to trauma has formed the basis for the view that local fluid loss is the predominating factor. As we have pointed out, the anæmic limb is also an anæsthetic limb, or at the very least a limb in which nervous reflexes are no longer normal. Again we would refer to the experiment in which shock was produced in an animal when the limb was excluded from its circulation but a second animal served to maintain its vascular supply and so its reflexes. In this case operation of the nervous factor alone produced shock as rapidly and as completely as did the operation of both factors together in the control animals. We have found it impossible to devise a converse experiment entirely free from logical objections, but in Experiment 17 (see Table II) this object was in some measure achieved. Despite a maximum fluid loss (the largest ever observed in our series of 100 experiments), the onset of shock was delayed for nine hours by the induction of spinal anæsthesia. While we must admit that this measure has possibly an effect on the humoral as well as on the nervous side, the practical applicability of the evidence is not impaired by any ambiguity with regard to its interpretation.

Work is still proceeding on the treatment of traumatic shock, and here we shall only outline the direction in which we consider that the accepted modes of therapy should be modified as a result of the conclusions reached in this paper. Our clinical experience, our experimental observations, and our study of the literature have convinced us that attempts to compensate for the fluid loss by means of intravenous therapy are largely ineffective. Up to the present, apart from the perfunctory administration of morphia, too little attention has been paid to the nervous side. The body itself possesses ample reserves of fluid, and the shocked case who recovers does so in large measure because he has been able to replenish a diminished volume by drawing on these natural resources. We have suggestive evidence to show that it is the continuance of abnormal nervous impulses which prevents this natural reaction on the part of the organism, and that if these impulses be controlled this favourable reaction takes place. The one therapeutic measure which succeeded in raising and maintaining the blood-pressure of a traumatized cat whose pressure had sunk to 20 mm of Hg was the induction of spinal anæsthesia. In some cases this measure has been combined with injection of saline into the peritoneum, with apparently beneficial results. It is of course possible that some better method of controlling nociceptive impulses may be devised—the injection of a local anæsthetic into the traumatized area suggests itself as one possibility—but we are convinced that control in some form is essential to the successful treatment of the syndrome. Even if we are wrong in the emphasis we lay upon the discharge of nociceptive impulses, the beneficial effect of spinal anæsthesia remains a fact which seems worthy of note and further investigation.

This work was carried out at the Buckston Browne Research Farm under the direction of the Master, Sir Arthur Keith, to whom we are indebted for constant advice and encouragement. The work followed on some earlier experiments made by one of us under the direction of Professor R. J. S. McDowall, whose advice has

been invaluable. In all the experiments described in this paper, Mr F Watson gave us most willing and valuable technical assistance.

We wish to make grateful acknowledgement for criticism of the draft manuscript to Sir Henry Dale and Professor P W D Wilkie.

BIBLIOGRAPHY

- BLALOCK, A, *Arch of Surg*, 1930, **xx**, 959, *Internat Clinics*, 1933 (Series 43), **i**, 144, *Surg Gynecol and Obst*, 1934, **lxi**, 551.
- BOLTEN, G C, ref *Zentralorgan f d ges Chirurgie*, 1925, **xxx**, 443.
- CANNON, W B, and BAYLISS, W M, *Lancet*, 1919, **i**, 256.
- CANNON, W B, *Traumatic Shock*, 1923. New York: Appleton.
- COENEN, H, *Mittheil med Woch*, 1926, **lxxvii**, **i** and 66.
- CRILL, G W, *Surgical Shock*, 1899. Philadelphia: Lippincott, *A Physical Interpretation of Shock*, 1921. New York.
- DALL, H H, LAIDLAW, P P, and RICHARDS, A N, *Med Research Council Reports*, 1919, **viii**, 8, *Jour of Physiol*, 1910, **xli**, 318.
- DALE, H H, and LAIDLAW P P, *Jour of Physiol*, 1919, **li**, 355.
- DALE, H H, *Bull Johns Hopkins Hosp*, 1933, **liii**, 397.
- EWIG, W, and KLOTZ, L, *Klin Woch*, 1932, **xxii**, 932.
- FELDBERG, W, and SCHILI, E, *Histamin*, 1930. Berlin: Springer.
- FREEDLANDER, S O, and LEVYHART, C H, *Arch of Surg*, 1932, **xxv**, 693.
- HOLT, R L, and MACDONALD, A D, *Brit Med Jour*, 1934, **i**, 1070.
- KALALOVA, V, ref *Zentralorgan f d ges Chirurgie*, 1925, **xxv**, 271.
- MCDOWALL, R J S, *Brit Med Jour*, 1933, **i**, 690.
- McIVER, M A, and HAGGART, W W, *Surg Gynecol and Obst*, 1923, **xxxvi**, 542.
- MELTZER, S J, *Arch of Internal Med*, 1908, **i**, 571.
- O'SHAUGHNESSY, L, *Burlston Browne Essay*, *Harveian Society of London*, 1931. Unpublished.
- PADGETT, E C, and ORR, T G, *Surg Gynecol and Obst*, 1928, **xlvi**, 783.
- PARSONS, E, and PHARMISTER, D B, *Ibid*, 1930, **li**, 196.
- REHN, E, *Langenbeek*, 1933, **clxxvii**, 360.
- RICH, A R, *Jour of Exper Med*, 1921, **xxviii**, 287.
- SCHNEIDER, H, *Deut Zeits f Chir*, 1930, **ccxxvii**, 343.
- SIMONART, A, *Arch Int de Pharmacodyn*, 1930, **xxxviii**, 269.
- SMITH, M I, *Jour of Pharmacol and Exper Therapeut*, 1928, **xxvii**, 465, 1928, **xxviii**, 239.
- THORPE, W V, *Biochem Jour*, 1928, **xxii**, 94.
- VOEGELIN, C, and DYER, H, *Jour of Pharmacol*, 1925, **xxv**, 102.
- WEISS, S, ROBB, C P, and ELLIS, C B, *Arch of Internal Med*, 1932, **xlvi**, 360.

SHORT NOTES OF RARE OR OBSCURE CASES

A CASE OF A TRUE HERMAPHRODITE

BY HAROLD MORISON, DRYDEN, ONTARIO

'HERMAPHRODITE' is a broad term used to include a fairly large group of individuals in whom the development of the external genitals is such that it is difficult or impossible to determine to which sex the individual belongs. The term, generally speaking, includes two classes of pseudo-hermaphrodite, the androgynous and the gynandrous. These two groups, the one having testicles with certain female characteristics in the external genitals, the other having ovaries with certain male characteristics, include 95 per cent of all cases of so-called hermaphrodites.

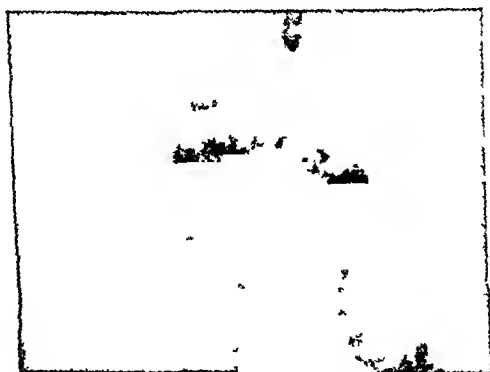


FIG 426 —Showing distribution of pubic hair



FIG 427 —External genitalia. Note large clitoris protruding from vagina

The true hermaphrodite is the individual who possesses, like the others, characteristics of the male and female on examination of the external genitals, but differs from the pseudo cases in that the individual has both ovarian and testicular tissues. The case here reported is, I consider, a case of a true hermaphrodite —

A N, Canadian born of Polish descent, age 26 years, labourer, came to me on Dec 27, 1933, complaining of a severe pain in his right lower abdomen, and vomiting. After a careful examination I decided that this individual had acute appendicitis. He was subjected to operation and an acutely inflamed appendix was removed.

During the course of the examination certain points of interest were noted —

- 1 The voice was soft, somewhat effeminate
- 2 The features were characteristically male, a two-day growth of heavy black beard was noted
- 3 The chest was quite flat, the nipples small
- 4 The abdomen was flat, narrow waist tapering below to a broad pelvis,

definitely female in type. The distribution of the pubic hair was typical of the female (Fig 426)

5 Examination of the external genitals (Fig 427) elicited the following points of interest. A vagina, from the upper part of which protruded a large penis-like clitoris $1\frac{1}{2}$ in in length. No scrotum or testicles were seen. The labia did not appear to contain any glandular tissue which might have suggested the presence of ovaries. There was no urethral opening in the enlarged clitoris. On raising the clitoris, however, one found a urethral opening at the junction of the base of the organ, and a groove which represented the vaginal orifice.

6 The legs were definitely female in type, heavy thighs, well-shaped calf and ankle.

It was decided to remove the appendix through a right rectus incision in order that a thorough examination of the pelvis could be made. On opening the abdomen I found on the left side of the pelvis a definite ovarian formation fixed in the pelvis. The intestines were picked off and retracted to procure a satisfactory view of the structure. It was white and nodular, no definite Graffian follicles could be seen. My assistants and I were quite certain that the structure was a fibrosed ovary. There was no broad ligament on the left side. In mid-pelvis there was no organ between the rectum and the bladder. I at first concluded that there was no uterine body present. In the right side of the pelvis, however, I found a mass of tissue the size of an orange, apparently of muscular formation, attached to what appeared to be a rudimentary broad ligament. This mass, which was very freely movable, was brought up through the incision and easily removed. I concluded that this mass was a uterus.



FIG 428—Photograph of the mass
4/3 6 cm removed from right pelvis.
The cut pedicle is at the bottom.



FIG 429—Section near base of mass showing
dense fibrous capsule above and a few subjacent glands
in looser connective tissue ($\times 30$) (Azo carmine
stain).

On careful questioning the patient stated that he had never experienced anything suggestive of menstruation, nor had he ever experienced sexual excitement or desire.

The specimen removed was sent to Dr Daniel Nicholson, Assistant Professor of Pathology, University of Manitoba, for examination. The following is his report—

The ovoid structure removed is shown in *Fig 428* and measures $4 \times 3 \times 6$ cm. Its large size, its shape, and smooth peritoneal covering gave the impression that it was a uterus. Serial transverse incisions showed the tissues to be very firm and especially fibrous subjacent to the peritoneal surface. This excessive amount of connective tissues is confirmed by the microscopic examination of sections stained by azo-carmine, which stains connective tissues a deep blue and muscle and glandular tissues a pale purple (*Fig 429*).

No evidence of uterine endometrium could be made out, but at widely separated intervals are islets of glands with masses of granular epithelioid cells adjoining them. These glands have the appearance of seminiferous tubules. Some of the cells lining the acini are definitely Sertoli cells, others which have no nucleoli



FIG 430—Two seminiferous tubules surrounded by a large aggregation of interstitial cells ($\times 110$) (Eosin and hæmatoxylin stain)

may be spermatogonia. The islets of granular epithelioid cells are the interstitial cells and they are present in greater numbers than in the average normal testis (*Fig 430*). The increase of interstitial cells has been frequently observed in cryptorchids.

In sections taken from near the base of the mass there are some irregular-shaped ducts lined by dark-staining columnar cells. These are the rudimentary structures of the rete testis. Both Professor R. G. Inkster and Professor J. L. Jackson of the Department of Anatomy identified these structures as being rete testis.

Summary—Large undescended testis containing a few areas of seminiferous tubules, an excess of interstitial cells, and much fibrous tissue. Operative findings combined with pathological examination of tissues removed places this case in the category of a true hermaphrodite.

PSOAS SPASM CAUSED BY A FOREIGN BODY

By T G ILLTYD JAMES, LONDON

THIS case is recorded on account of the unusual features it presented

R C, an ill-nourished and neglected infant of 2 years, was admitted to hospital with a foreign body in his throat. There was no respiratory or œsophageal obstruction, but a skiagram revealed the presence of a large object in the pharynx, together with an assortment of foreign bodies in the abdomen. A large brooch was removed without difficulty from the pharynx, while the other objects were allowed to be expelled naturally—the usual treatment being administered. The needle in *Fig 431* (which shows the whole collection) was the last to be passed—seven days after admission, its course being watched by frequent skiagrams. The child was then discharged, none the worse for the experience.

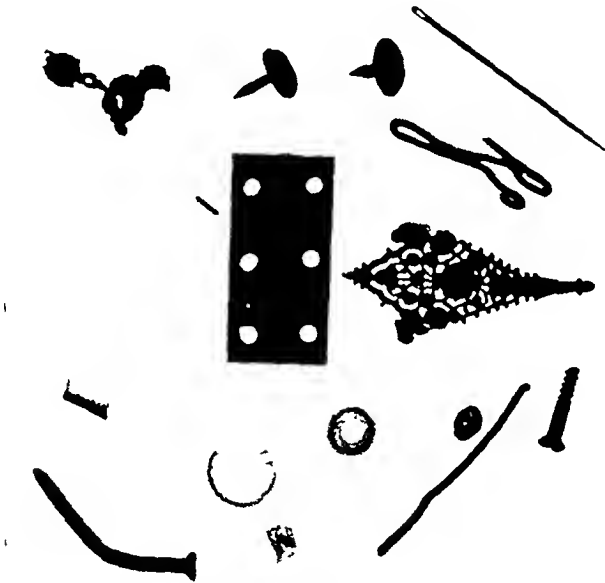


FIG 431—Foreign bodies passed naturally from the abdomen of a boy of 2 years

Seven months later the boy was re-admitted for pain in upper part of right thigh and flexion deformity of right hip. The hip was acutely flexed, and attempts to extend it were painful. Further flexion and rotatory movements were free from pain. No other abnormality was discovered, and constitutional signs were absent.

A skiagram revealed a normal hip and spine, but showed the needle depicted in *Fig 432*. Screening with a barium meal indicated that the needle was partly in the muscles of the posterior abdominal wall and partly in the duodenum. Laparotomy confirmed this diagnosis. The point of the needle could be felt in the transverse portion of the duodenum, with the greater part embedded deep in the right psoas muscle. The needle point was made to protrude through the

anterior surface of the duodenum. The most interesting feature of the whole case now presented itself, for the points of two needles protruded, in close apposition to one another. The needles were extracted, and the tiny hole was closed by a purse-string suture.

All pain and deformity at the hip disappeared after the operation, and convalescence was uneventful.



FIG 432—Skiagram showing position of needle

COMMENTS

1 Perforation of the posterior surface of the duodenum by sharp foreign bodies which have been ingested is unusual. Psoas spasm from this cause must be very infrequent.

2 The complete superimposition of two needles of equal size, point to point and eye to eye, appearing in the skiagram as one, is a unique feature of this case.

ACUTE INTUSSUSCEPTION IN AN ADULT

By C H CUFF

SURGICAL SPECIALIST, CYPRUS

AND M GOSDEN

GOVERNMENT BACTERIOLOGIST, CYPRUS

ACUTE intussusception in the adult is a comparatively rare occurrence, and it is thought that there are unusual features in the following case which make it worth recording.

HISTORY—The patient was a Greek woman aged 45. In March, 1932, she came to the Government Hospital complaining of a raised pigmented tumour just external to and below the right outer canthus. She stated that there had been since birth a small black mole in this situation, and that three months previously she had accidentally scratched it, since when it had enlarged rapidly.

On examination she presented a coal-black conical-shaped tumour approximately 2×2.5 cm just below the right outer canthus and commencing to involve the lower lid (*Fig 433*). A diagnosis of melanoma was made, and as she refused an operation the growth was treated by surface application of radium. A total dose of 1008 mgrm-hours was given. She was seen again in July, 1932, when the growth had disappeared, leaving a smooth flat scar (*Fig 434*). She was heard of from time to time as being quite well until April, 1934, when she



FIG 433—Melanoma of the face



FIG 434—Appearance after treatment

was admitted to hospital again with classical signs of acute intestinal obstruction. The duration of the symptoms before admission was thirty hours. She stated that six weeks previously she had had a similar attack which had passed off after twelve hours.

ON EXAMINATION—The abdomen was soft and somewhat distended and visible peristalsis was marked. In the right iliac region a mobile indefinite tumour was palpable. P R and P V nil. Faecal vomiting present.

OPERATION (April 10, 1934)—Mid-line subumbilical incision. The colon was found collapsed and empty, the jejunum was distended and intussuscepted about 8 ft from the duodenal junction. It was found quite impossible to reduce this intussusception, and the affected gut was resected. Numerous pigmented deposits were seen in the mesentery and omentum. The patient did well for a few weeks and then succumbed to metastases.

EXAMINATION OF SPECIMEN—The specimen consisted of a piece of jejunum 30 cm long with a portion firmly intussuscepted into it. The walls of both the inner and outer layer were swollen and purple, no actual gangrene was present.

The apex of the intussusception consisted of a firm jet-black nodule 2 cm in diameter. A similar smaller nodule was present in the wall 14 cm from the apex (*Fig 435*). Section of the nodule showed a typical secondary melanotic sarcoma in the wall (*Fig 436*).



FIG 435 —Portion of intussuscepted intestine

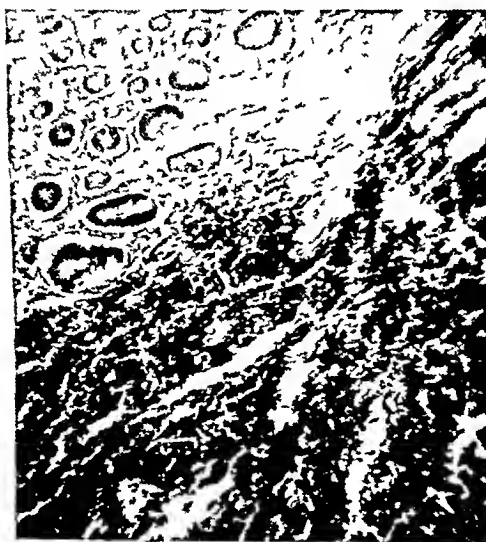


FIG 436 —Section of growth in intestine

SUMMARY

A case of melanotic sarcoma of the face is described, in which complete freedom from symptoms for two years followed treatment by radium. Acute intussusception occurred from secondary deposits in the small intestine.

MIXED TUMOUR ARISING IN THE PALATE

BY T FRANCIS JARMAN

LATE ASSISTANT MEDICAL SUPERINTENDENT, ROYAL SEA-BATHING HOSPITAL,
MARGATE

THIS case is presented as being of rare occurrence and of very considerable interest both from clinical and pathological points of view

A male patient, aged 17, was admitted to the Royal Sea Bathing Hospital suffering from abdominal tuberculosis. In the course of a routine physical examination a tumour of the palate was discovered. The patient was quite unaware of the presence of the tumour, had no idea of the date of its origin, and had, indeed, never been conscious of the presence of any abnormality in connection with the palate. The tumour was about the size of a walnut and situated in the centre of the soft palate, it was very firm in consistency, and though it appeared to be somewhat adherent on its deep surface to the hard palate and was not freely movable, it did not give the impression of actually arising from the hard palate.

OPERATION—On May 30, 1933, an operation for removal of the tumour was performed. It proved to be extremely easy of removal and was enucleated without any difficulty. There was little bleeding, and the incision in the palatal mucous membrane was lightly sutured with fine catgut. Healing occurred rapidly by first intention.

PATHOLOGICAL FINDINGS—The tumour was about the size of a walnut, of a slightly lobulated and irregular spheroidal shape, very firm in consistency, and surrounded by a fairly thick well-formed fibrous capsule.

On section the cut surface was smooth, dense, and of a white colour, throughout which were scattered a few small yellowish areas of a hyaline appearance.

Microscopic examination of a section of the tumour showed that it was surrounded by a well-formed fibrous capsule and that it consisted in part of cells with darkly staining angular nuclei, which cells were arranged in some places in irregular branching columns and in others showed a tendency towards duct and acini formation, and in part of areas showing degenerative changes and metaplasia resulting in the formation of myxomatous material and pseudo-cartilage (*Figs 437, 438*).

The tumour thus appeared to be exactly comparable with the so-called mixed tumours, the origin of which is more commonly associated with the parotid and submaxillary salivary glands.

SUBSEQUENT PROGRESS—Communication was made with the patient on May 10, 1934, in reply to which he volunteered information to the effect that the palatal wound remained soundly healed, that there was no evidence of any lumps whatsoever at the site of removal of the tumour or anywhere else in the mouth, and that no lumps or swellings were evident in the neck or elsewhere—sufficiently definite and reliable information on which to conclude that so far, after the lapse of one year from the time of removal of the tumour, there is no evidence of the presence of any recurrence.

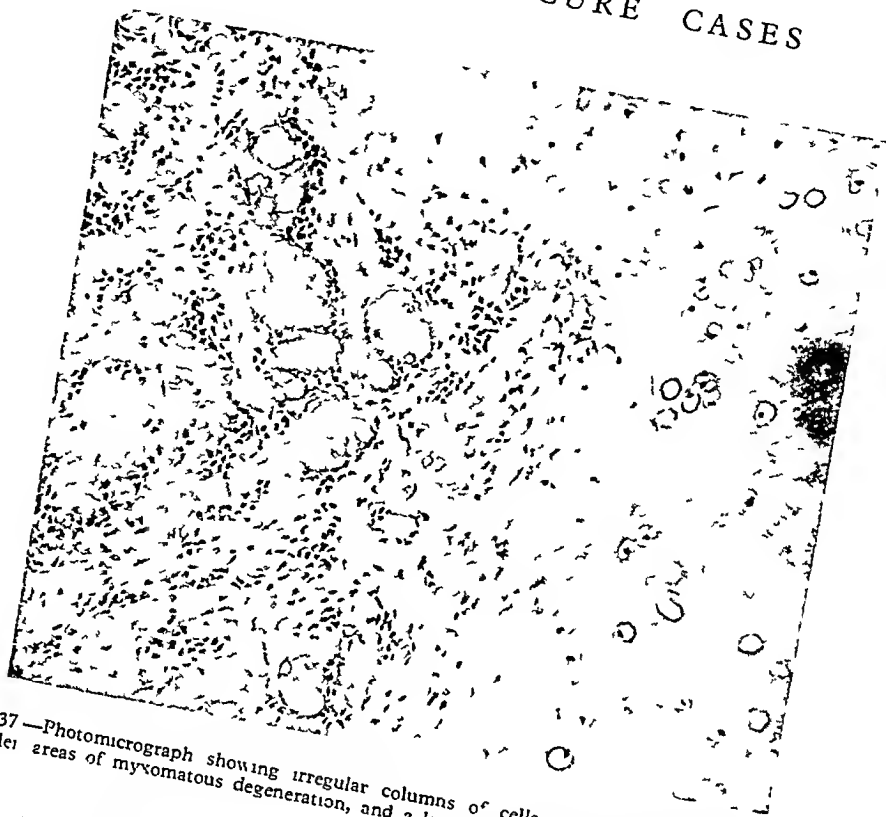


FIG 437—Photomicrograph showing irregular columns of cells with darkly staining angular nuclei; areas of myxomatous degeneration, and a large area of pseudo cartilage ($\times 90$)

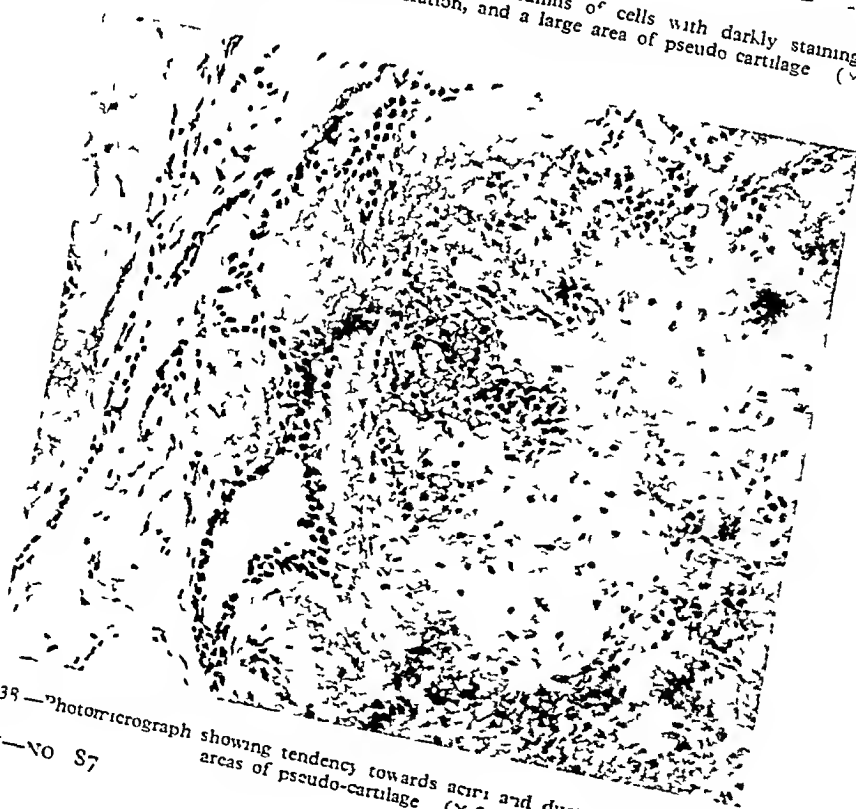


FIG 438—Photomicrograph showing tendency towards acini and duct formation, and some areas of pseudo-cartilage ($\times 90$)

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COMMENTS

Tumours of the so-called mixed type, the origin of which is usually associated with the salivary glands, are of extremely rare occurrence in the palate. D H Patey,¹ in an authoritative and exhaustive article, considers these 'mixed' tumours of the salivary glands from a pathological point of view; he found that of the 55 cases of mixed salivary tumours which he collected, 5 occurred in the palate. Of these 5, 4 have remained free from recurrence for some years and 1 developed a local recurrence two years after removal of the primary tumour.

R Sonnenschein² has reported two cases and has reviewed the literature up to 1929. He states that mixed tumours of the soft palate are of comparative rarity, well within fifty cases. Of the two cases which he reports, one had an enormous growth in the soft palate which, when removed, looked like a uterine fibroid, and on section and microscopic examination showed "epithelial sheets resembling carcinomatous tissue, myxomatous tissue, alveoli filled with colloid, epithelial pearls, etc." Other cases are reported by Beck,³ Heitz,⁴ Malan,⁵ and Carruthers.⁶ Beck's case presented a firm growth in the right side of the palate, extending into the supertonsillar and post-nasal space. Biopsy revealed a "myxo-fibro-chondroma"; enucleation was performed, but recurrence took place three weeks later, which recurrence was successfully treated by electro-coagulation. Professor Malan describes one of his two cases as a "myxo-lympho-angio-endothelioma" and the other as almost identical with this except that it presented also a "pseudo-sarcomatous zone". The histological appearance of the case reported by Carruthers is identical with that now presented.

The origin and pathological nature of these tumours has been widely discussed, and it has been suggested that those occurring in the palate arise from embryonic salivary rests. M Creysel⁷ regards these palatal tumours as arising from already existing salivary gland tissue, and considers also that the polymorphism is more apparent than real, being the result of interaction between an epithelial and connective tissue. D H Patey,¹ to whose excellent article all those seeking detailed information and discussion with regard to the pathological nature of these mixed tumours might well be referred, concludes that "they are a composite group of epithelial tumours the varying pathological features of which depend on the degree of differentiation attained, rapidity of division of the cells, and the amount of myxomatous change undergone by the epithelium."

REFERENCES

- ¹ PATEY, D H, *Brit Jour Surg*, 1930, Oct, 241
- ² SONNENSCHN, R, *Trans Amer Laryngol Assoc*, li, 235, *Arch Otolaryngol*, 1930 Feb, 137
- ³ BECK, J C, *Trans Amer Laryngol Rhinol and Otol Soc*, 1930, xxvi, 385
- ⁴ HEITZ, M, *Lyon med*, 1933, July 9, 28
- ⁵ MALAN, A, *Arch ital di Laring (Supp)*, li, 11
- ⁶ CARRUTHERS, D G, *Med Jour Australia*, 1932, Dec 3, 23
- CREYSEL, M, In discussion on the case of M Heitz (q v)

PHLEGMONOUS GASTRITIS

BY C JENNINGS MARSHALL, LONDON

HISTORY—W T, a man of 54 years, was admitted to hospital for pneumonia on Dec 12, 1932. Empyema was identified by puncture of the left pleural cavity on Jan 11, 1933, it was drained by rib resection under local anæsthesia on Jan 13. The organism was a pneumococcus, the reaction was of a very adynamic type, the temperature for ten days preceding drainage not exceeding 99°.

The recovery from the pleural infection was uneventful until Feb 15, the tube having been removed and only a short sinus into the chest remaining, on that date the patient began to vomit, no food was retained and vomiting persisted in the absence of its administration. On Feb 17 the patient looked ill and emaciated, though not profoundly so. The mind was clear, and the complexion and lips were clean, the tongue was heavily furred and dry. At intervals of an hour or so a very striking vomitus was produced, it was a copious bright red jelly, transparent, and not admixed with any recognizable formed element. There was no pus, no bile, no stercoraceous material. The jelly was amphoteric to litmus.

ON EXAMINATION—The temperature was subnormal (and remained so both before, and for some seven days after the time of this examination). Physical signs were conspicuous by their absence. The pulse was 104, the respiration 20. The abdomen was flat, without visible peristalsis, there was resistance by the upper part of the recti, but without any notable tenderness. An enema produced flatus and a small faecal result.

DIAGNOSIS—The nature of the vomit was obviously the clue to the case, absence of bile or other intestinal content showed it to be gastric, the mucus and blood without trace of acid secretion would be accounted for by an acute inflammation of the mucosa abolishing its function. Recollection of published cases of phlegmonous gastritis, however, recalled a far more desperate picture than the one present—in particular contrasting with the complete absence of pain in association with the vomit. The comparatively afebrile character of the empyema could be set off against this, and it was thought best to explore.

EXPLORATION—The stomach was diffusely and intensely œdematous and rigid, the swelling extending from cardia to pylorus and also into the gastro-hepatic omentum. The retroperitoneal tissues were œdematous and the glands enlarged. The thickness of the gastric wall was estimated at about $\frac{1}{2}$ in. The tissues were glistening and semi-translucent. Close inspection in places revealed a milkiness suggestive of leucocytic infiltration, but nowhere was there a localized pus collection. The peritoneal coat was smooth and shiny, there was no perigastric lymph deposit and no rolling up of omentum. No trace of gastric ulcer could be seen. The liver and gall-bladder were normal. There being clearly nothing to be done, the abdomen was closed.

AFTER-HISTORY—Following this 'demonstration in force', the patient made a complete and rapid recovery. Tided over by rectal saline, oral feeding was resumed on the fourth day after operation. The mucoid vomit gradually ceased, at no stage being accompanied by pus. The ultimate recovery was free from dyspeptic symptoms, which had not been present before the illness either.

COMMENTS

The term 'phlegmon' derivatively refers to a 'burning' or inflammatory swelling, and therefore, suppuration not being an essential in the definition, this instance of acute cellulitis of the gastric walls which resolved without formation of pus is an important addition to the clinical picture of the disease. In the literature the difficulty of diagnosis has always been insisted on, here it was clear enough, the only difficulty being the incompleteness of the published description of clinical variations.

As regards the pathogenesis, consideration for the progress of the patient obviously precluded opening the stomach to make further investigations. Diffuse and local forms of acute pyogenic gastritis are well recognized, associated with gastric ulcer, trauma, or carcinoma, there may be local or diffuse abscess formation, sometimes with perforative peritonitis, liver abscess, or empyema. In the case under consideration the presumption arises that this was an instance of pneumococcal phlegmon of the stomach, probably hæmic in origin.

SARCOMA GROWING IN A CALCIFIED ADENOMA OF THE THYROID

By H W L MOLESWORTH, FOLKESTONE

THE following case appears worthy of publication on account of its rarity

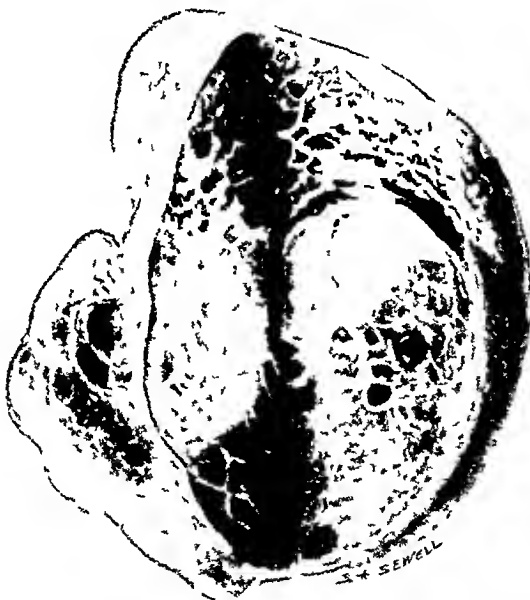


FIG 439—Calcified adenoma of the thyroid gland (R C S, 7222 2)

A male patient, aged 59, complained of a swelling in the left side of his neck for one week. There was a hard nodule in the left lobe of the thyroid, about the

middle, which was not adherent to the surrounding structures, and there was no evidence of nerve involvement. At operation the whole of the left lobe of the thyroid was removed. The specimen was sent to the Royal College of Surgeons



FIG 440 —Microscopical section of the tumour ($\times 80$)

Museum, and on examination proved to be a calcified adenoma of the thyroid from the capsule of which a spindle-celled sarcoma had developed (*Fig 439*). *Fig 440* is a microscopical section of the tumour. The patient remains well at the time of writing, one year after the operation.

REVIEWS AND NOTICES OF BOOKS

Surgery of a General Practice By ARTHUR E. HERTZLER, M.D., Chief Surgeon, Halstead Hospital, etc., and VICTOR E. CHESKY, M.D., Chief Resident Surgeon, Halstead Hospital. Large 8vo. Pp. 602, with 427 illustrations. 1934. London: Henry Kimpton. 42s. net.

THE authors of this book, who are surgeons and not general practitioners, admit in their preface that they do not know what should be included in minor surgery, i.e., the surgery needful for a general practitioner, but they state that the scope of the book embraces such diseases and injuries as may be treated in the practitioner's office or the patient's home. The actual subject matter, however, far exceeds this definition according to the standards of this country.

Where true minor surgery is dealt with, i.e., such conditions as local injuries and infections, skin tumours, wound treatment, and the like, the methods advocated are on the whole satisfactory. It should be remarked, however, that no mention is made of the injection treatment of piles or of 'closed' drainage of empyema. Further, the suture of a fistula in ano after excision is advocated as a routine—this method, though valuable at times, is surely not advisable in the hands of a general practitioner.

Where more serious conditions are considered, such as carcinoma of the breast, tongue, and cervix, the advice given is crude and unsatisfying. The statement in reference to carcinoma of the cervix that "palliative measures—the cautery or acetone—may be used when a radiologist is not available or the patient does not have the price" is one which it is difficult to take seriously.

The Newcastle upon Tyne School of Medicine, 1834-1934 By G. GREY TURNER, assisted by W. D. ARNISON. Large 8vo. Pp. 224 + vii. Illustrated. 1934. Newcastle upon Tyne: Andrew Reid & Company Ltd. 10s. net.

THIS history may well serve as a model for other schools of medicine which from small beginnings as local proprietary teaching centres have developed into the medical faculty of one of the more recently founded universities. The Newcastle upon Tyne School of Medicine has been fortunate in its historians. Dr. Dennis Embleton published in 1890 *Collegium Medicum Novocastræ* and he joined the school five years after it was opened. Dr. Arnison became a student in 1882 and Professor Grey Turner in 1895. Each is able to speak from first-hand knowledge. The present writers summarize Dr. Embleton's history and carry it on until the present centenary year. Like St. Cuthbert, the school was itinerant for many years, moving from house to house until it seems at last to have found a permanent resting place with fully equipped buildings. It began from the very smallest beginnings, yet it soon secured recognition by the great licensing bodies at a time when such recognition was jealously guarded. The high standard of surgery which it quickly reached was due to the existence of the City Infirmary, where George Yeoman Heath, Rutherford Morison, and Grey Turner in succession found plenty of material upon which to teach, whilst the accidents and diseases incident to a rapidly increasing industrial population enabled them to attain a high standard of operative skill. Of all this the authors tell in a pleasant manner, and illustrate what they have to say with a wealth of pictures of the men and places they speak of. They also add a plan of the sites occupied at different times by the school of medicine. They provide also for the general reader two amusing and interesting chapters on life in the school in 1882-6, one written by Dr. Arnison, the other by Professor Grey Turner as he knew it several years later. All is now different. The type of student has changed, the eccentrics have vanished, but it is pleasant to have their memories recalled by those who knew them personally. As verbal corrections for the new edition which is sure to be wanted, it may be noted that Sir William Fergusson is uniformly spelt Ferguson and that Professor F. W. Andrewes was not Andrews.

The Science and Practice of Surgery By W H C ROMANIS, M A, M B, M Ch (Cantab), F R C S (Eng), F R S (Edin), Surgeon and Lecturer on Surgery, St Thomas's Hospital, etc., and PHILIP H MITCHNER, M D, M S (Lond), F R C S (Eng), Hon Surgeon to H M the King, etc. Fifth edition. Royal 8vo. In two volumes. Vol I, General Surgery. Pp 789 + 75 pp of index. Vol II, Regional Surgery. Pp 962 + 75 pp of index. 758 illustrations. 1934. London. J & A Churchill. 14s each volume.

THE fifth edition follows post-haste on the fourth. It is less than two years since it was our duty to review the latter, and what we then said might, with equal truth, be said of the present edition. Most of the chapters on the specialities or on special methods of investigation are up to date and modern in their outlook. The chapter on certain regions of general surgery are lamentably out of date—thus the sympathetic nervous system still receives scant mention, as does the present-day wire traction in the treatment of fractures, yet there is still room for the description of amputations which neither author can have ever seen performed. Has not the treatment of malignant disease by mass radiation made sufficient progress to be worthy of mention? It might with profit replace at least four out of the five methods of removing the tongue which have no present-day application.

We would again urge the necessity of re-writing certain chapters in view of recent progress, and the advantage to the publication which would result from the omission of all out-of-date methods of treatment.

Surgery of the Thorax By T HOLMES SELLORS, M Ch, M A, B M (Oxon), F R C S, Assistant Surgeon to Queen Mary's Hospital for the East End, etc. Large 8vo. Pp 519 + xxiii, with 140 illustrations. 1933. London. Constable & Co Ltd. 22s 6d net.

THE necessity for a book on surgery of the thorax of reasonable size is unquestioned, and this fact affords sufficient justification for this review of the subject. Thoracic surgery has undergone such enormous developments during the last fifteen to twenty years that it is quite impossible in a volume of this size to do more than give a brief outline of the subject, but its value is increased by a good bibliography. The chapter on the physiology, which contains all the essentials of this subject, will repay reading by all those who perform even the occasional chest operation. Necessarily, there must be points which are debatable and with which the reviewer may not be in agreement, but there is such a complete lack of dogmatism that he is somewhat disarmed. In fact, for the surgeon who does occasional thoracic operations, it might be advisable if the author were more critical and dogmatic instead of offering a variety of procedures, many of which are of historical, and occasionally of minor historical, interest only. There is a very strong flavour of Continental surgery in the advocacy of extensive resections of the bony chest wall for certain types of thoracotomy. Many of these are surely unnecessary, and except where the chest wall is actually involved by neoplasms, restoration of the chest wall to as near normal as possible should be the ideal to be aimed at in so far as it compatible with efficient approach to the intrathoracic structures. Chapters are included on the heart, œsophagus, diaphragm, and aneurysm, apart from those devoted to pulmonary and mediastinal diseases.

The whole volume shows evidence of wide reading, is well written and printed, and the illustrations are admirably reproduced, and should form an excellent basis for those interested in the surgical side of chest disease.

Cirugía gástrica By Dr MANUEL CORACHAN (Barcelona). Vol I. Crown 4to. Pp 775 + xiv, with 374 illustrations. 1934. Barcelona. Salvat Editores, S A. No price given.

THE full value of this book cannot be appraised until the whole work is available, but much may be said about this present handsome volume. In the prologue Dr Ribas Ribas, who also writes the introduction for his former pupil, the author, says that "at one stride, awakening (incipiente) Spanish Medical Literature attains the full height of the best foreign literature in quality if not in quantity." There is truth in what he says, but to those who know what Barcelona could produce in the way of books almost a hundred years ago (witness, for example, the history of the world in forty lavishly illustrated volumes published in 1845) this splendid piece of bookmaking will come as no surprise. The typography is excellent, the illustrations

are very good, and the binding is sound. Moreover, the contents deserve such form. This volume deals with preliminaries and clinical features, treatment is reserved for Volume II, so that the opportunity for appreciating the author's direct contribution to gastric surgery is so far restricted. Nevertheless, amidst a very complete exposition of contemporary opinion, Dr Corrichan's personality peeps out continually and with effect. After chapters dealing with anatomy, physiology, and embryology, the clinical examination of the 'gastro-duodenal' patient is taken and full accounts are provided not only of radiology (with beautiful pictures of the normal and abnormal mucosal patterns), but also of gastroscopy and gastro-photography.

In the chapter on the etiology and evolution of gastric ulcer, it is noteworthy that the author considers that ulcer-cancer is a not infrequent sequence. The later chapters deal with simple and specific gastritis, with tumours innocent and malignant, with gastroparesis, with gastric crises, pyloric spasm, volvulus, diaphragmatic hernia, diverticulosis, invagination, fistula, and injuries.

We look forward to the opportunity of reviewing the treatment volume which will complete a great work.

Localization of Function in the Cerebral Cortex By Various Authors Vol XIII of a Series of Research Publications Editorial Board S T ORTON, M D, J F FULTON, M D, T K, DAVIS, M D Medium 8vo Pp 667 + xxi, with 171 illustrations 1934 Baltimore The Williams & Wilkins Company (London Baillière Tindall & Cox) 36s net

THIS book constitutes Volume XIII of the annual reports of the meetings of the Association for Research in Nervous and Mental Diseases, and includes the papers presented at the thirteenth annual meeting of the Association held in New York on Dec 28-29, 1932.

The contents may be divided into three sections. The first is devoted to the anatomy and comparative ontogeny of the cerebral cortex. The second deals with the physiological studies of the function of the cerebral cortex. The third section, which consists of the major part of the book, is concerned with clinical studies of a varied and wide nature.

It is impossible to review all the twenty-one chapters which go to make up this book, but some are of outstanding interest and importance.

Dr Charles Frazier and Dr Stuart Rowe contribute a most interesting and practical chapter on certain observations upon the localization in fifty-one verified tumours of the temporal lobe. These writers found that tumours in this so-called 'silent' area gave cortical localizing symptoms in 63 per cent of cases, whilst uncinate attacks were very uncommon. Another most interesting fact which emerges from this chapter is that over half the tumours of the left temporal lobe failed to produce aphasia.

Drs Wilder Penfield and Joseph Evans have written a chapter on "Functional Defects Produced by Cerebral Lobectomies." These authors come to the conclusion that cerebral lobectomies, even though involving large amounts of brain substance, are followed by surprisingly little disturbance of function which can be detected by ordinary methods of examination.

The illustrations are very good, in fact they are themselves quite a feature of the book. The type is clear, and the format and general 'make-up' are excellent. The book will prove of value to the physiologist, neurologist, and neuro-surgeon alike, as well as to the anatomist who is interested in the cerebral cortex.

Actinotherapy Technique An Outline of Indications and Methods for the Use of Modern Light Therapy With a Foreword by Sir HENRY GAUVAIN M D, M Chir (Camb), F R C S Crown 8vo Pp 184 Slough The Sollux Publishing Co 6s net

THIS small book serves a double purpose. In the first place it gives a brief account of the physical basis and physiological principle of 'light treatment' followed by an alphabetical list of the various conditions in which the method is useful, together with technical details for its application. Secondly, there are references to nearly 1000 books and periodicals which give first-hand descriptions of the scope and application of actinotherapy. It certainly forms an invaluable guide for the student and practitioner.

Die parasagittalen Meningiome By Dr H OLIVECRONA (Stockholm) 10 $\frac{1}{2}$ x 7 $\frac{1}{4}$ in Pp 144, with 145 illustrations 1934 Leipzig Georg Thieme Paper covers, M 24, bound, M 26

DR OLIVECRONA has done a real service to neurosurgery by the publication of this monograph on the parasagittal endotheliomata (meningiomas) of the cerebral hemisphere. The author has followed Cushing's lead in applying the term 'parasagittal' to those growths arising from the angle between the falx cerebri and the superior longitudinal sinus. Of the author's series of 82 cases of meningioma, 34 belong to the category under consideration. The clinical phenomena are discussed according to the exact site of origin of the tumour, whether from the anterior, middle, or posterior third of the longitudinal sinus, or from the lateral aspect of the falx cerebri below the sinus. A chapter deals with the morbid anatomy of these growths, but no detailed study is attempted of the histogenesis. There were 5 deaths out of the 33 cases submitted to operation, the causes being traceable to secondary infection associated with a cerebrospinal fistula (1 case), and pulmonary complications (2 cases). A further 10 per cent of the patients died after an interval. More or less complete cure occurred in half the patients, while in a quarter there was a residual defect limiting their capacity for work. This monograph can be thoroughly recommended as a necessary addition to the library of every surgeon interested in neurology.

The Clinical Management of Horseshoe Kidney By ROBERT GUTIERREZ, A B, M D, F A C S, Chief of Clinic of the Department of Urology, James Buchanan Brady Foundation of the New York Hospital, etc. With a Foreword by Dr EDMOND PAPIN (Paris) 10 $\frac{1}{2}$ x 7 $\frac{1}{4}$ in Pp 143 + xiv, with 52 illustrations 1934 New York Paul B Hoeber Inc \$3 00

THIS monograph, the contents of which originally appeared in serial form in the *American Journal of Surgery*, contains a complete account of what is known on the subject of that congenital deformity called 'horseshoe kidney'.

As the result of his study of 25 cases the author has come to the conclusion that this condition is something more than just a malformation, he considers that its mere existence constitutes a disease, the 'horseshoe kidney disease', this owns its characteristic signs and symptoms, and to obtain relief from these it is necessary to perform the operation of symphysiotomy, division of the isthmus uniting the two organs.

It is of interest to note that no fewer than 19 of the 25 cases were diagnosed before operation, of the remainder, 2 were discovered at operation and 4 at autopsy. This great improvement on the figures published by former authors must have been brought about by the recent advances in urography.

The author has devised a triangle to be used as an aid in the interpretation of skiagrams, its value is based on the fact that the lowest calices of the two kidneys are always much nearer to one another than in normally situated kidneys.

After reading the book with genuine interest the reviewer thinks that there are several statements which are open to criticism.

In spite of the author's assertion that the mere existence of a horseshoe kidney is sufficient to give rise to characteristic symptoms it seems obvious, on reading the case histories, that every one of the 19 cases was suffering from some complication—infection, stone, hydro-nephrosis, or neoplasm. If the pain due to the fixity and 'incarceration' of the isthmus were of any severity, one would have expected the mean age of the patients to be something less than 30, the youngest was 18, and the majority were between 40 and 50.

It is difficult to follow the author when he states that the pressure of the isthmus, situated at the level of the bifurcation of the aorta, exerts a baleful influence on the coeliac plexus and the receptaculum chyli, and is it even certain that pressure on the sympathetic does cause pain?

Though Dr Gutierrez is such an enthusiastic advocate of the operation of symphysiotomy as a cure for 'horseshoe kidney disease', it does not appear from this book that he has ever done one, two of the cases were treated, quite correctly, by heminephrectomy, and the others by dilatation of the ureters and lavage of the renal pelvis.

The illustrations are beautifully reproduced, and the skiagrams are clear and helpful, there is a good and not too extensive bibliography and an index.

Le Genou Anatomie chirurgicale et radiographique Chirurgie opératoire By Professor ANTOINE BASSER, Surgeon to the Beaujon Hospital, Paris Royal 8vo Pp 189, with 120 illustrations 1932 Paris Masson et Cie Fr 45

THIS small monograph on the anatomy and surgery of the knee-joint is comprehensive and well illustrated. The account of the anatomy both from the surface and by dissection is supplemented by radiographs of the normal joint in various positions and by a number of diagrams indicating the lines of all the important tendons and ligaments superimposed over those of the bones. These are drawn with great care and accuracy so as to make them of great value for reference. Another series of X-rays is given after barium injection of the synovial cavity and the arteries. About twenty different lines of incision for surgical approach are figured and described, together with the various methods of dividing the patella.

In the section dealing with fractures involving the joint, although the statement is made that it is desirable to reduce the number and size of foreign bodies used in osteosynthesis practised near a joint to the minimum, most of the illustrations given show that this principle has been grossly violated.

Most of the usual methods for suture of a fractured patella are described and illustrated, but there is no mention of the use of a pedicled strip of fascia lata as a suture material. In the operations for meniscotomy, the French method of making a long transverse incision dividing the lateral ligament is given a prominent place, without due reference to the danger that this method involves of weakening the stability of the joint. The repair of the cruciate ligaments and the different methods of arthrodesis and of arthroplasty are well described and illustrated.

The clearness of the illustrations, especially in the anatomical section, is one of the best features of the book.

Chirurgische Tuberkulose By Dr MAX FLISCH-THEBESIUS, Senior Medical Officer of the Surgical Section of the Private Hospital, Frankfurt a M. With an Introductory Note by Prof Dr V SCHMIEDEN. Medium 8vo Pp 194 + iv, with 58 illustrations 1933 Dresden and Leipzig Theodor Steinkopff Paper covers, R M 15, bound, R M 16 20

THIS small book is an excellent summary of the subject of surgical tuberculosis. From first to last it is essentially practical, as befits a volume in the series of 'Practical Medicine', of which it forms the fifteenth number.

In regard to the problem of diagnosis, the author takes very much the same view that is held in this country—that is to say, whilst acknowledging the value of various so-called specific tests, he does so with considerable reservation. Animal injection and biopsy each has a certain though restricted role in diagnosis.

Essentially the book is a description of the modern methods of treatment of surgical tuberculosis by the conservative methods of rest, fresh air, and light. Various methods of surgical fixation, e.g., Albee's bone-graft for the spine and different types of arthrodesis, are mentioned as having a limited application, but there is no description of their technique.

Manipulative Treatment for the Medical Practitioner By T MARLIN, M D, Ch B D P H, R C P S (Eng.), D M R E, Medical Officer in Charge of the Massage, Electrotherapeutic, and Light Departments, University College Hospital, etc. Demy 8vo Pp 133 + vii, with 86 illustrations 1934 London Edward Arnold & Co 10s 6d net

MANIPULATIVE treatment is usually interpreted by the medical writer as including movement of joints through their normal range, and stretching of ligaments and of muscles. The other manipulations of muscles and soft tissues are the work rather of the masseur. Therefore the more usual surgical manipulations are based upon a known pathology, adhesions are broken down, shortened muscles and ligaments are stretched. Even in the case of manipulations for a locked knee or temporo-mandibular joint we have a fairly clear idea of what is happening to the intra-articular cartilage. But the manipulations of the osteopath are in many cases based upon a much less definite and much more obscure pathology. Mr Marlin has attempted the difficult task of describing and explaining manipulations of this class, with, it is to be feared, a result that is only partially successful. It is perhaps a pity that the greater

part of the book is devoted to the technique of manipulation, with only one chapter at the end on the choice of chronic cases for such treatment

It is to be feared that the very wide difference between the medical and the osteopathic views of pathology makes it impossible to explain osteopathic methods in medical terms, but at least it is possible to study the clinical details in cases that are helped by manipulations as a guide to the prescription of treatment in other cases Mr Marlin's book fails to supply much that is still required, but nevertheless it does approach the subject of manipulative treatment from a new angle, it gives many useful details of methods, and it will therefore necessarily be read by all who are interested in surgical manipulations

Infections of the Hand A Guide to the Surgical Treatment of Acute and Chronic Suppurative Processes in the Fingers, Hand, and Forearm By ALLEN B KANAVEL, M D, Sc D, Professor of Surgery, Northwestern University Medical School, Chicago
Sixth edition Pp 552 + xvi, with 216 illustrations 1934 London Bailliere, Tindall & Cox 30s net

It is twenty years since Kanavel began his studies on this subject, and the fact that his book has now reached its sixth edition proves that his teaching has been widely diffused His two outstanding contributions to the correct understanding and treatment of infections of the hand have been first, the distinction between lymphangitis and suppurative tenosynovitis, and second, the accurate description of the fascial spaces in the palm which are distinct from those of the tendon-sheaths Both these teachings are now universally accepted The present edition contains much new matter such as that relating to infections from bites and injuries from the teeth, the pathology and treatment of metacarpo-phalangeal joint infections, of gangrenous infections, of injuries from indelible pencils, of cattle-hair and other peculiar infections Chapters have been added upon the function of the hand, the use of splints, and the prophylactic treatment of injuries One of the outstanding features of this, as of the former editions, is the beauty and clearness of the illustrations

Röntgendiagnostik der Knochen- und Gelenkkrankheiten By Prof Dr ROBERT KIENBOCK (Vienna) Part 3 Gelenksosteomatose und Chondromatose Large 8vo
Pp 228, with 194 illustrations 1934 Berlin and Vienna Urban & Schwarzenberg RM 22 50

THE third part of this series deals with osteophytes, cartilaginous and bony loose bodies in the joints, and malignant cartilaginous and bony tumours of joints It consists largely of cases illustrated by radiographs and is more adequately illustrated than the previous numbers It constitutes quite a good atlas of the subject, but unfortunately the diagnosis given is frequently open to question and subsequent operative findings are not always given

Lord Lister the Discoverer of Antiseptic Surgery By C J S THOMPSON, M B E, Hon Curator of the Historical Collection of the Museum of the Royal College of Surgeons of England Crown 8vo Pp 99 Illustrated 1934 London John Bale, Sons & Danielsson, Ltd 5s net

THIS is a short account of the life and work of Lord Lister, written in a popular style suitable for the general public It certainly fulfils its object of giving a brief account of the greatest of the movements of surgical evolution and of the personality of its hero in an interesting fashion readily understandable by the ordinary reader Lister's modest character, his indomitable energy, and his steady progress to ultimate achievement and triumph are well told, so that the reader will probably go on to the perusal of the larger and more complete life by Godlee

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers]

- Surgical Applied Anatomy** By SIR FREDERICK TREVES, Bart, revised by C C CHOYCE, C M G, C B E, B Sc, M D, F R C S, Professor of Surgery, University of London, etc. Ninth edition. F'cap 8vo. Pp 720, with 174 illustrations, including 66 in colour. 1934. London. Cassell & Co Ltd. 14s net.
- Chirurgie du Pancréas** By P BROcq (Paris) and G MIGINIAC (Toulouse). Large 8vo. Pp 428, with 74 illustrations. 1934. Paris. Masson et Cie. Fr 75.
- Head Injuries** By L BATHE RAWLING, M B, M Ch (Cantab), F R C S, Consulting Surgeon to St Bartholomew's Hospital and to the West End Hospital for Nervous Diseases. Demy 8vo. Pp 86 + vi, with 22 illustrations. 1934. London. Oxford University Press. 7s 6d net.
- Illustrations of Regional Anatomy** By E B JAMIESON, M D, Senior Demonstrator and Lecturer, Anatomy Department, University, Edinburgh. F'cap 4to. Published in five sections. Section I, Central Nervous System, 48 plates, 7s 6d. Section II, Head and Neck, 61 plates, 10s. Section III, Abdomen, 37 plates, 5s 6d. Section IV, Pelvis, 30 plates, 3s 6d. Section V, Thorax, 27 plates. 1934. Edinburgh. E & S Livingstone. Complete set, 30s net.
- The Anatomy of Surgical Approaches** By L C KELLOGG, A B, M D, Professor of Anatomy, College of Medical Evangelists, Loma Linda and Los Angeles, California. Crown 8vo. Pp 134 + v, with 29 illustrations. 1934. London. Bailliere, Tindall & Cox. 7s net.
- L'Epaule. Anatomie des Formes extérieures, Anatomie radiographique, Chirurgie opératoire** By ANTOINE BASSET, Professeur agrégé à la Faculté de Paris, and JACQUES MIALARET, Interne des Hôpitaux de Paris. Royal 8vo. Pp 292, with 116 illustrations. 1934. Paris. Masson et Cie. Fr 65.
- Diseases of the Rectum and Colon and their Surgical Treatment** By J P Lockhart-Mummery, M A, M B, B C Cantab, F R C S, Senior Surgeon to St Mark's Hospital. Second edition. Large 8vo. Pp 616 + viii, with 247 illustrations. 1934. London. Bailliere, Tindall & Cox. 35s net.
- Modern Operative Surgery** Edited by G GREY TURNER, M S, F R C S, F A C S (Hon.), Senior Surgeon, Royal Infirmary, Newcastle-upon-Tyne, etc. Second edition. In two volumes. Medium 8vo. Pp 1764, with 860 illustrations in text and 19 plates. 1934. London. Cassell & Co Ltd. £3 3s net.
- Diseases of Children** Edited by HUGH THURSFIELD, D M (Oxon), M A, F R C P, Physician, Hospital for Sick Children, Great Ormond Street, Consulting Physician, St Bartholomew's Hospital, and DONALD PATERSON, M D (Edin), F R C P, Physician to Out-patients, Hospital for Sick Children, Great Ormond Street, Physician in Charge of Diseases of Children, Westminster Hospital. With contributions by 36 authors. Third edition. Royal 8vo. Pp 1152 + xii, with 277 illustrations. 1934. London. Edward Arnold & Co. 50s net.
- A Synopsis of Surgical Anatomy** By ALEXANDER LEE MCGREGOR, M Ch (Edin), F R C S (Eng), Lecturer on Surgical Anatomy, University of the Witwatersrand, Assistant Surgeon, Johannesburg General Hospital. With a Foreword by Sir HAROLD J STILES, K B E, F R C S (Edin). Second Edition. Crown 8vo. Pp 644 + xx, with 639 illustrations. 1934. Bristol. John Wright & Sons Ltd. 17s 6d net.
- Applied Anatomy the Construction of the Human Body considered in Relation to its Functions, Diseases and Injuries** By GWILYM G DAVIS, M D, Late Professor of Orthopedic Surgery and Associate Professor of Applied Anatomy in the University of Pennsylvania. Revised by GEORGE P MULLER, M D, Professor of Clinical Surgery, Graduate School of Medicine, University of Pennsylvania, and five Associates. Ninth edition. Large 8vo. Pp 717 + xii, with 674 illustrations. 1934. London. J B Lippincott Company. 42s net.
- The Cure of Hæmorrhoids and Varicose Veins** By STUART MCAUSLAND, B A (Lond), M D, CH B (Liv), M R C S, L R C P, Hon. Physician, Hahnemann Hospital, Liverpool, etc. Second edition. Demy 8vo. Pp 77 + x, illustrated. 1934. London. John Bale, Sons & Danielsson Ltd. 4s net.

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IPSISSIMA VERBA

BY SIR D'ARCY POWER, KBE, LONDON

V THE FIRST SUCCESSFUL OPERATION FOR GALL-STONES IN ENGLAND

A SUCCESSFUL operation for gall-stones was reported on June 15, 1867, by Dr Bobbs in the *Indiana Journal of State Medical Science* for 1868 (p 63), and another by Drs Musser and Keen in the *Transactions of the College of Physicians of Philadelphia* (1884, Vol VII, Series III, p 265). It was not until Mr Lawson Tait read "A Case of Cholecystostomy" at a meeting of the Royal Medical and Chirurgical Society on Nov 11, 1879, that an English surgeon could report a successful case. He says (*Med-Chir Trans*, 1880, LXIII, 16) —

"Dr Marion Sims attributes to Dr Handfield Jones, and I think correctly, the merit of first suggesting that the liver and gall-bladder should be included within the field of surgical practice and to my good fortune it has fallen to be the first to follow out Dr Handfield Jones' idea and Dr Sims' plan successfully.

"Elizabeth M — aet 40 was admitted to the (Birmingham) Hospital (for Women) on August 18th 1879 on account of an abdominal tumour.

"She had been married eighteen years, had borne six children, her menstruation had always been normal and she had enjoyed perfectly good health until the summer of 1878. At this time she began to suffer severe spasmodic pains in the right side, these being always aggravated by walking or by lifting even slight weights. In September she noticed swelling at the seat of pain and this slowly increased. During last winter her pain became much more intense, her appetite failed, she lost strength and flesh rapidly and on admission she presented an emaciated and almost cachectic appearance. She also suffered at that time from incessant headache and sickness and obstinate constipation. The seat of pain was over the right kidney where there was a heart-shaped tumour, firm and elastic, in which no fluctuation could be detected, and which was extremely tender to the touch. On examination under ether this tumour was found to be perfectly moveable towards each side, indeed, it could be pushed completely across the middle line to the left side. All round it a note of intestinal resonance could be produced. When pushed over to the left side, its heart-like shape became very apparent, and when it lay on the left side of the vertebral column, with its apex directed downwards and to the left, its base evidently retained a connection with the right side.

"A careful examination of the urine gave only negative results, though she spoke vaguely of its having been occasionally dark in colour, muddy and deficient in quantity

"At the consultation held with my colleague Dr Edginton upon the case, a variety of suggestions were made for diagnosis, the chief of which were cystic enlargement of a floating kidney, a tumour of the head of the pancreas, and dropsy of the gall-bladder. But no decided diagnosis was attempted and my proposal to open the abdomen and thus ascertain the nature of the tumour was agreed upon

"On August 23rd (1879) I opened the abdomen in the middle line, to the extent of four inches, the umbilicus forming the centre of the incision. It then became at once evident that the tumour was a distended gall-bladder. I passed the needle of an aspirator into the apex and drew off a quantity of white starchy-looking fluid, probably amounting to between twelve and fifteen ounces but I cannot speak positively as to its amount, as it was unfortunately thrown away by a nurse immediately after the operation. I then opened the gall-bladder at the point of puncture, so as to admit my finger and came at once upon a large round gall-stone lying loose in the cavity. This I easily removed and on further search I found another of rather larger size, and probably of pear-shape, at the entrance of the duct, impacted in it and evidently the cause of the dropsical distension of the gall-bladder. The removal of this stone was a matter of very great difficulty, in fact it took a very much longer time to effect than all the other steps of the operation put together. From the long narrow funnel-like cavity in which it was lodged and from the mobility of the bladder it was very difficult to seize and when at last I did get hold of it I found it adherent to the mucous surface. I had then to consider the extreme likelihood that in removing this impacted stone I might tear the walls to which it was attached and thus certainly kill my patient. I therefore performed a very careful and protracted lithotomy, chipping little fragments off the stone regularly all over its exposed surface till I had the satisfaction of lifting out its nucleus. I then passed a blade of a pair of fine forceps on each side of it and by a gentle squeeze broke up the remainder and was then enabled to lift it all out. The weight of the stone removed entire is 4.2 grammes and that of the fragments I could gather of the broken stone is 2.9 grammes, but of the latter stone as much again must have been lost on the sponges which were packed into the wound during the process of crushing and upon which I had constantly to wipe my instruments. I washed out the cavity repeatedly and took every precaution that I could to secure that no fragments were left. I then stitched the wound in the gall-bladder to the upper end of the wound in the abdominal walls by continuous sutures, leaving the aperture into the bladder quite open and then I closed the rest of the abdominal opening in the usual way. The operation was performed with complete antiseptic precautions and the anæsthetic employed was ether

"She rallied from the operation completely in a few hours. I dressed the wound antiseptically the same evening at 11 p.m. and found the dressings stained with healthy bile. In the further progress of the case there is very little to report save that the flow of bile from the wound continued till the 3rd of September when the dressings were discontinued and zinc ointment was used in their place. The stitches were removed and the wound was completely healed on September 9th when she began to take solid food, up to that time her diet being restricted to milk and beef tea. On the 14th she sat up for the first time and on the 30th she went

home quite restored to health, free from pain and all her former symptoms and having gained at least a stone in weight

"Looking back upon this case I do not think that a more accurate diagnosis was possible. Fortunately our advanced practice in abdominal surgery makes our



MR LAWSON TAIT

limited powers of diagnosis in such a case of less importance and I thoroughly agree with Dr Sims that we should not wait until the approach of almost fatal symptoms puts the diagnosis in unmistakable fashion but that 'we shall make an early exploratory incision ascertain the true nature of the disease and then carry out the surgical treatment that the necessities of the case may demand' "

PROGRESSIVE POST-OPERATIVE GANGRENE OF SKIN

By A M STEWART-WALLACE

MEDICAL FIRST ASSISTANT AND REGISTRAR, LONDON HOSPITAL

IN the London Hospital this year, under Dr Robert Hutchison, there occurred, following a thoracotomy and drainage of an empyema, a case of a remarkable and rare post-operative complication of a slowly progressive gangrenous infection of the skin and subcutaneous tissues. This gradually spread in spite of all therapeutic measures, till, after eight months, it stretched from the occiput to the iliac crest behind and extended anteriorly to involve the whole abdominal surface, and finally caused the death of the patient. Several physicians, surgeons, and others at the London Hospital saw the case, but none of them could remember having met with this remarkable condition before. It was, in fact, only subsequently, quite by chance, that the present writer came upon a similar case in an American journal. This prompted a search into the literature, with a result that it was found that over thirty cases have been reported in America during the last ten years and that the condition has been discussed at at least three Surgical Society meetings, cases also have occurred in Germany, Italy, and Australia. It was realized, firstly, that the condition is not generally known in this country, one case recorded by Hector Scotson³² last year, which included also a brief summary of eight cases collected from the literature, is the only report that can be found in English journals*. Secondly, and of greater importance, it was realized that failure to recognize the condition may lead to failure, as in our case, to carry out the only effective treatment—namely, excision of the advancing edges, an operation which in almost all the published cases lead to rapid healing of the lesion.

For these reasons it is felt that this case should be recorded and a review included of all the published cases and literature that throw light on this remarkable condition.

THE CONDITION AS AN 'ENTITY'

A study of the cases, 37 of which have been collected in all, at once convinces one that they must be grouped together as an entity and that they constitute a rare post-operative infection of the skin with characteristic and peculiar features demanding a clearly defined line of treatment.

Cullen¹ in 1924 reported a case of "progressive enlarging ulcer of the abdominal wall", which measured 12 in across, following drainage of an appendicular abscess. It failed to respond to all therapeutic measures, but rapid healing was

* Interest in this condition is increased by the appearance, subsequent to preparing this paper, of a report of a case, only the second to be recorded in this country, in the current number of the BRITISH JOURNAL OF SURGERY (1934, LXXI, 392) by Nightingale and Bowden in which they describe progressive gangrene of the skin following laparotomy for a perforated duodenal ulcer. The infection spread steadily till it covered an area 3 × 6 in, when the advancing edges were excised and rapid healing resulted.

eventually brought about by excision of the margins and applying pinch skin-grafts. In the same year Christopher² described a case of "severe spreading carbuncular infection of the chest wall" following rib resection and drainage of an empyema. Soreness about the wound developed on the third day, red areas round the stitch-holes on the sixth, and in the third week large pieces of necrotic tissue were removed, the process spread, accompanied by intolerable pain in spite of various local applications, light therapy, and blood transfusions, though the sinus healed up. Eventually after three months it extended from the scapular spine to the iliac crest behind, and anteriorly nearly reached the umbilicus. The advance of the lesion at this stage was successfully checked by excision of the edges, necessitating ten separate debridements under general anaesthesia, and rapid healing resulted.

In 1926 Brewer and Meleney^{4, 5} described two cases of "progressive gangrenous infection of the skin" following drainage of appendicular abscesses, each was eventually checked by surgical measures. They recognized that these cases were similar in nature to those of Cullen and Christopher, and Meleney carried out some animal inoculation experiments on the etiology which will be referred to later. The cases were discussed before the American Surgical Association in Detroit,⁷ where five similar cases were briefly described by other surgeons present.

After this there appeared at intervals several reports of cases, the majority following drainage of appendix abscesses, all of which were recognized by their authors as being similar in nature to the cases described. All were slowly progressive, carbuncular-like or gangrenous in appearance, with serpiginous outline. They were very painful and peculiarly intractable, being resistant to all ordinary therapeutic measures. The sloughs came away, leaving the base covered with red granulation tissue, which in places showed a tendency to heal, with the appearance of islands of epithelial cells, and the ulcerating undermined edges distinct and separate from the original sinus, which may still be draining or be a healed scar. In nearly all the cases excision and cauterizing of the advancing margins, sometimes in several stages, led to healing, this taking from three to twenty-two months from the date of the original operation to be completed.

At a meeting of the American Medical Association, Philadelphia, in 1931 Lynn²⁴ read a paper on the subject and included a fresh case of his own. Among those present who joined the discussion were Cullen and Shipley, who previously had published cases, and also Meleney, who had had the unique experience of being able to study and experiment with two typical cases. It was agreed that the similarity between the cases justified them being regarded as a clinical entity and that all the evidence was in favour of a common pathological and etiological basis. The descriptions of twelve further cases which have occurred since then only tend to confirm this conclusion.

INCIDENCE

Although there are only 37 cases which we have been able to collect in the literature, it is stated by some of the authors that the condition is more common than these few literary contributions would lead us to believe. Thus both Ballin²⁷ and Meleney³⁷ state that oral communication with other surgeons shows that most of them have seen one or more of these cases without recognizing their importance.

But on the other hand, Meleney's cases^{41, 48} of "hæmolytic streptococcal gangrene" which will be discussed later must not be included in this group. It is also difficult to be convinced that one of Ballin's cases,²⁷ that of an intractable ulcer following an inguinal gland abscess, is of the same type. He excludes climatic bubo in this case by the absence of Leishman-Donovan bodies, but it has recently been conclusively shown that this condition is due to a filter-passing virus.⁴⁴ McDonagh,³⁸ in a letter commenting on Scotson's case,³² says he has had experience of several cases, some following appendicectomy, some occurring spontaneously, and some also in the genito-crural region, that they are due to *B. morganii* and *B. gangrenæ cutis*, and that they respond to novarsobenzene injections. It will be seen that these cases must be different from the series described here. Before including cases it is necessary to make certain that they showed the remarkably intractable and slowly progressive features which characterize all the cases in the series under consideration. It is no doubt true that many unsuccessfully treated cases have not been recorded and that cases have been reported, owing to the absence of a generic term to describe them, under titles which have not been consulted in our search of the literature.

That the condition is in fact very rare is suggested by the evidence that, though of course abscess formation and cellulitis of the abdominal wall is relatively not an infrequent post-operative complication, gangrene of the chest or abdominal wall is remarkably uncommon in the records of the London Hospital. Thus in the period 1903-33 (excluding cellulitis, gangrene following extravasation of urine, and scrotal gangrene from peri-urethral abscesses) only eight cases have occurred in the chest or abdominal wall which have been described in the notes at any stage as "gangrenous" in appearance. Five of these were acute infections of the tissues round the wound leading to abscess formation, or cellulitis with sloughing and "gangrene" of the overlying tissues (four after drainage of appendicular abscesses, one after abdomino-perineal excision of a rectal carcinoma). One was a case of gas gangrene of the abdominal wall following incision of an ischio-rectal abscess secondary to an advanced carcinoma of the rectum, and one followed removal of a stone and drainage of the gall-bladder in a patient with persistent glycosuria in which sloughing of the whole thickness of the abdominal wall took place. In these thirty years one case only can be found in the records of the London Hospital which may have the same etiology as the condition discussed, it is briefly described here. It is not proposed to include it in the series, as the available evidence is not sufficiently conclusive.

J. H. was operated on in 1925 for a radical cure of an inguinal hernia. "A remarkable gangrene, organism unknown, developed in the wound and spread to involve an area two inches in diameter, and sloughing of the skin and subcutaneous tissues occurred." There are no further notes except that the temperature chart showed a low-grade fever and a pulse-rate averaging 110 to 120 for one month after the operation, on which date the patient left the hospital cured.

NOMENCLATURE

Many different names have been used to describe this condition. Most of the cases during the last three years, including that of Scotson, the first to be reported in this country, have been described under the title "Progressive Post-operative Gangrene of the Skin", a term first used by Brewer and Meleney⁴ in 1924. This

expresses the three characteristics of the lesion and the present case is reported under that heading. Now that the condition has been accepted as an entity it is highly important that some definite term should be universally adopted to describe it, in order to facilitate references and to discriminate it from other skin infections.

CASE REPORT

HISTORY—A male, aged 44, was admitted to the London Hospital under Dr Robert Hutchison on Jan 20, 1934, with the history of an illness which had commenced with a cold and cough nine weeks previously. One week later pain developed on the left side of his chest, sharp in character and made worse by breathing deeply and coughing. The same day he coughed up a little blood, but since then his sputum remained clear. He stayed in bed with the cough, chest pain, and pyrexia for eight weeks, three times having attempted

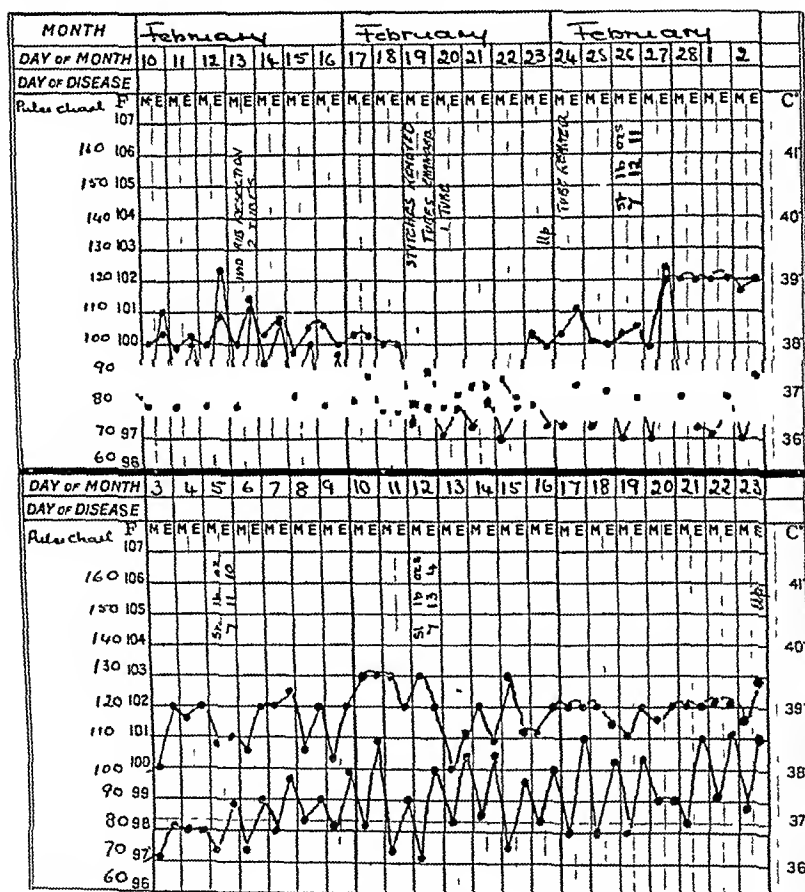


FIG 441—Temperature and pulse chart in a case of progressive post-operative gangrene of the skin

to get up, but being driven back to bed on each occasion by an increase of the symptoms. He was eventually sent up to hospital as his home doctor had found signs of left-sided pleural effusion. Three sputum tests had been reported negative for tubercle bacilli.

Previous Illnesses—Influenza in 1919. No chest complications. No other illnesses remembered. No history of jaundice. Denied taking alcohol.

Family History—Two brothers and one sister had acholuric jaundice (investigated by Dr Janet Vaughan). One brother was well following splenectomy, the other two died

following splenectomy Father, mother, six other brothers, two other sisters, and four children showed no signs of acholuric jaundice

ON EXAMINATION—The patient was found to have an intermittent pyrexia 100° to 102° in the evening and 98° to 99° in the morning Pulse 100 to 110, respirations 20 He was a thin man, with a good colour, no jaundice, definite clubbing of fingers, heart and arteries normal except for a short systolic apical murmur, blood-pressure 120/80 There was flattening of his left apex, with hollowing above and below the clavicle, and diminished movement of the whole of the left side of his chest His heart apical impulse was $1\frac{1}{2}$ in internal to the nipple line and he had a dull percussion note, absent breath-sounds, and tactile vocal fremitus at the left base extending up nearly to the spine of the scapula The left apex and right lung were clear except for a few transitory crepitations at the right apex



FIG. 442.—The lesion on the fifty-ninth day antero lateral view

Nothing abnormal was found in the abdomen or central nervous system Urine normal No tubercle bacilli found in the sputum

Blood-count (Jan 22)—Red cells, 3,400,000 Hæmoglobin, 61 per cent Colour index, 0.9 Leucocytes, 12,400 Polymorphs, 83 per cent No eosinophils Slight poikilocytosis and anisocytosis Moderate polychromatophilia

Van den Bergh—Direct, negative Indirect, less than 0.2 mgrm per cent Fragility of red cells very slightly increased Complete hæmolysis to 0.35 per cent Partial hæmolysis to 0.45 per cent Control's blood partial hæmolysis to 0.3 to 0.4 per cent

Jan 24—Paracentesis of chest, 16 oz of thick opaque grey-green pus aspirated, a film of which showed degenerate pus and many Gram-positive cocci in chains Culture grew non-hæmolytic streptococci No tubercle bacilli

Feb 13—X-ray of chest Heart and mediastinum displaced to the right Left lung showed opacity of the middle and lower zones consistent with the presence of fluid Right lung, clear

OPERATION—A swinging temperature persisted till on Feb 13 a thoracotomy was performed under local infiltration with 1 per cent novocain. Two inches of the left eighth rib were removed subperiosteally between the posterior axillary and the mid-scapular lines. Two pints of thick pus were evacuated, two tubes inserted, and the tissues stitched closely round to set up a closed drainage system. Empyema wound drained well. Washed out daily with Dakin's solution.

SUBSEQUENT PROGRESS—On Feb 19 the edges of the wound were red, inflamed, and bulging round the tubes, stitches and tubes were therefore removed, and one short large-bore tube was inserted and the wound allowed to drain into the dressing. Temperature and pulse-rate were settling well and the general condition was good. By Feb 20 (7th day after operation) the temperature and pulse-rate were normal and the wound was discharging very little, though the edges were œdematous, red and tender. By Feb 23 the patient



FIG 443 —The lesion on the fifty-ninth day postero lateral view

appeared so well that he was allowed up for a short period. His pulse-rate, however, began to rise, and on Feb 27 (14th day) it rose to 120 and was rarely below 110 after this. Meanwhile a swinging pyrexia insidiously started and gradually increased (Fig 441). The wound looked more angry, the œdema and redness began to extend into the surrounding skin, and the edges to slough. Necrosis of the central area took place, and by March 14 (29th day) the wound was described as resembling a large carbuncle. The actual sinus looked healthy and was producing only a little thick discharge. The black, leathery necrotic slough separated in several places, exposing the muscles, which were not affected at any stage and formed the centre of the "carbuncular process", the edges were tender, raised, serpiginous, and undermined, it was surrounded by a band of red œdematous tissue which was fairly sharply demarcated from the healthy skin. Later the necrotic area separated, leaving the tender

raised margins fairly distinct from the central floor of red granulation tissue covering the muscle layer, which in places showed a tendency to heal

By March 27 (41st day) the wound stretched from the sternum anteriorly to the edge of the vertebral column posteriorly. The anterior and posterior extremities had acutely inflamed and advancing margins, while the intermediate edges were comparatively healthy and shelving, with outgrowths of epithelium spreading across the base of the ulcerated area. By April 14 (59th day) the process had reached the stage shown in Figs 442, 443, extending beyond the right nipple line anteriorly. The lesion then began to spread upwards and downwards, and slowly progressed till at the end of six months it involved the extensive

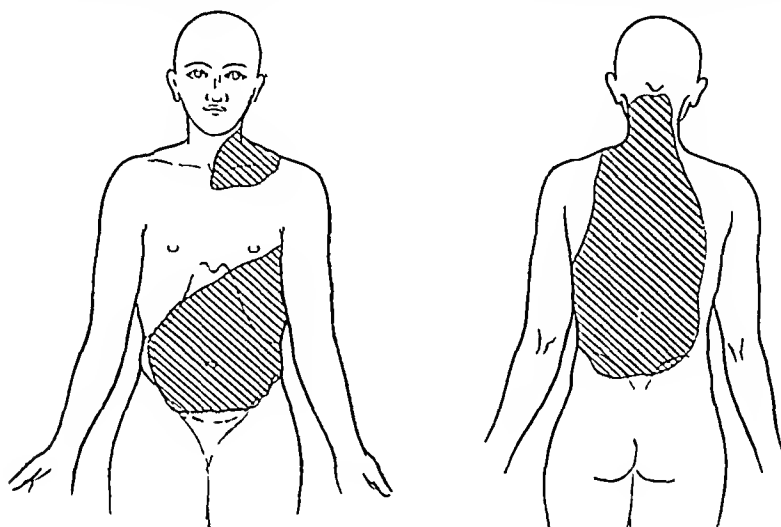


Fig 444 —Diagram to show extent of lesion at death (thirty second week)

area shown in Fig 444. The sinus had healed up and there were several islands of epithelium round the site. The edges were exquisitely tender, and great difficulty was experienced with the dressings and finding a comfortable position for the patient to lie in. Apart from pain on pressure and during the dressings there was little spontaneous pain, and sedatives were not required to the same extent as in Christopher's case. In himself the patient was not very ill, but as the process spread and involved such a wide area and the swinging pyrexia continued for so many months he began to show signs of wasting, and during the last two or three weeks he lost his morale and strength, and eventually died quietly on Aug 27, 194 days (32 weeks) after the operation. His appetite remained good almost to the end.

Blood-counts —

	March 27	April 3	June 27	July 20
Red cells	4,800,000	4,000,000	4,400,000	4,100,000
Hæmoglobin	77%	66%	60%	65%
Colour index	0.79	0.82	0.68	0.8
White cells	15,000	13,600	9,800	12,600
Polymorphonuclears	72%	75.5%	81%	79%
Eosinophils	2%	1.5%	2%	2.5%
Large lymphocytes	9%	9.5%	10.5%	10.5%
Small lymphocytes	11%	8%	2%	6%
Large hyaline	6%	5.5%	4.5%	2%
Basophil	0%	0%	0%	0%

Blood Culture —Sterile (May 4)

Blood Wassermann Reaction —Negative (June 12)

Radiograms of Chest —March 3 —“The condition at the left base is improving. The heart is returning to the left chest. The diaphragm is beginning to move. There is, however, an area of density in the mid-axillary line which might be fluid or might be thickening.”

May 20—"The chest shows no collection of pus now The whole condition is much clearer" (M H Jupe)

Culture Reports—April 7—Pus from sinus "Streptococci in direct film Pale and abundant growth of *Staphylococcus aureus*"

April 10—Non-hæmolytic streptococci No influenza bacillus (A Greaves)

April 12—Swab of wound No true diphtheria bacilli present Films and cultures show presence of *Staphylococcus aureus* in great abundance

Sputum—July 12—No tubercle bacilli Chiefly pneumococci with some staphylococci, sarcinæ, and *M catarrhalis*

TREATMENT—

General—High vitamin diet Iron, arsenic, strychnine, cod-liver oil, malt, and advita capsules were given by mouth at different stages Massage to legs

Local Applications—Various, including glycerin and magnesium sulphate, orthoform powders

Sedatives—Luminal, aspirin, pyramidon, and heroin

April 13—36,000 units of diphtheritic antitoxin

April 14 and 18—Staphylococcal toxoid (1/60) 0.1 cc and 0.3 cc respectively not repeated as pulse-rate and temperature appeared to be higher after the two injections

April 16—Light treatment started Appeared to make condition worse

June 21—Aug 10—Weekly course of autogenous staphylococcal vaccine No apparent effect

Blood Transfusions on July 26 and 31 and Aug 11 and 18 No improvement

CLINICAL FEATURES

Of the cases collected, 28 were male and 6 were female The age distribution is shown in the following table

AGE	CASES	AGE	CASES
18	1	50-60	5
30-40	9	60-65	8
40-50	3	70	1

The operations after which the infection occurred are tabulated below —

OPERATION	CASES	REFERENCES
Appendix abscess Drainage	20	Cullen, ¹ Brewer and Meleney, ^{4 5} Porter, ^{7 8} Moschovitch, ¹⁰ Clinton, ¹¹ Alexander, ¹² Mayeda, ¹³ Gordon, ¹⁶ Cole and Heide- man, ¹⁷ Meleney, ²³ Lynn, ²⁴ Warfield, ²⁵ Mitchell, ²⁶ Ballin and Morse, ²⁸ Baker and Terry, ³¹ Scotson, ³² Meleney, ³⁵ Carol ³⁶
Upper abdominal abscess Drainage (One probably appendicular, ¹⁴ one subhepatic following a previous perforated duodenal ulcer ⁴⁹)	3	Brewer and Meleney, ⁶ Shipley, ¹⁴ Meleney ³³
Simple appendectomy No drainage	1	Gillespie ¹⁵
Purulent cholecystitis Removal of gall-bladder	1	Porter ⁹
Chronic cholelithiasis Drainage of gall-bladder	1	Freeman ¹⁹
Carcinoma of cæcum Cæcostomy	1	Meleney ³⁴
Pelvic inflammation Oophorectomy and drainage	1	Borelli ³
Empyema Rib resection and drainage	6	Christopher, ² Poate, ²¹ Brunsting, ²² Ballin and Morse, ²⁷ Patterson, ³⁰ Stewart-Wallace ^{36a}
Suppurating epididymitis	1	Ballin and Morse ²⁹
Breast abscess sinus Drainage	1	Probststein and Seelig ¹⁸
Ventral hernia through old cholecystectomy incision Repair	1	Tennant ¹⁰

It will be seen that of the 37 cases 33 followed drainage of a purulent infection, of which 21 were appendix abscesses and 6 were empyemata

In about half of the cases the first sign of the new infection is apparent during the first week, and in the majority of the remainder during the second week, the extremes being the first²³ and twenty-second day³⁰ In the later group the pyrexia and pulse-rate usually return to normal after the operation and the condition of the patient appears completely satisfactory, in Patterson's case he was even sent home However, as the new infection gradually sets in, the temperature and pulse begin to rise again The infection starts as soreness, redness, and œdema about some small part of the wound, or more commonly about the stitch-holes, particularly of the tension sutures The process gradually spreads, and in the second or third week necrosis takes place in the centre, with formation of black leathery sloughs which then separate and leave a relatively healthy base covered with red granulation tissue In no single case were the muscles or any of the deeper tissues affected, and the original wound or sinus in most cases healed up, or if it continued to drain, as in most of the thoracotomy cases, the sinus itself looked very healthy

In places the edge shows a tendency to heal and becomes shelving and non-œdematous, and epithelium grows out across the base to meet small islands of deep epithelium arising from sweat glands or hair follicles that have not been destroyed The remainder of the edges are raised, undermined, and œdematous, and spread slowly⁶ (about 4 mm a day) but relentlessly, and unless checked by surgical measures enormous areas of the skin of the body are ulcerated The most extensive lesions were seen in the cases following empyemata, in Poate's case²¹ the whole back from the occiput to the middle of the buttocks on both sides was involved In several of the abdominal cases the entire abdomen was affected, while in addition in one case⁶ it extended to involve as well the whole anterior and lateral aspects of the thigh down to the knee, and in another⁹ the whole area between the right scapular spine and the iliac crest A marked feature is the exquisite tenderness of the active edges, which leads to great difficulty with dressings and in more than one case even required general anæsthesia for these to be carried out This great pain on the slightest manipulation was a feature on which particular stress was laid in twelve of the cases

Most cases were accompanied by an irregular temperature up to 99° or 100° and sometimes 101° in the evening (102° and 103° in the later stages in our case) and a raised pulse-rate averaging 100 to 120 Most patients suffered from exhaustion from pain and lack of sleep rather than toxæmia, and several records state that the appetite remained good throughout A moderate leucocytosis was found in some cases A full post-mortem on Poate's case²¹ showed no abnormalities of any viscus other than those usually associated with chronic empyemata Microscopic study of the edge of the lesion²³ shows extensive fragmentation of the dense subcuticular connective tissue and a heavy cellular infiltration of the subcutaneous fat There is no thrombosis of the vessels, which are universally dilated and filled with blood with a large number of polymorphs clinging to the walls This suggests that the gangrene is due to the direct action of some lytic substance on the tissues rather than a cutting off of the blood-supply Masses of Gram-positive cocci are seen in the centre of the lesion, and scattered organisms in diploform or short chains in the periphery

DIFFERENTIAL DIAGNOSIS

Before discussing the etiology of this interesting condition it is important, in order to avoid confusion, to differentiate and discriminate between "progressive gangrene" of the skin and other similar conditions —

1 Common wound infection which may go on to abscess formation and cellulitis and will respond usually to opening up the tissues. If the organism is particularly virulent or there is general or local lowering of resistance on the part of the patient, sloughing of the tissues and acute gangrene of the abdominal wall may occur as in a case fully described recently by Horsley⁴⁰. Before the days of aseptic surgery infectious gangrene was a frequent post-operative complication, and vivid descriptions of its horrors may be found in many of the older text-books.

2 Erysipelas, which, though it may resemble it on the first day, never leads to progressive ulceration.

3 Gas gangrene, with its much more severe symptoms, the prostration of the patient, the foul-smelling discharge, with crepitation of the surrounding oedematous, discoloured skin, and the positive bacterial findings of anaerobic gas bacilli. Recently a case confined to the skin and subcutaneous tissues has been described.⁵⁰

4 "Hæmolytic streptococcal gangrene" of the skin. Meleney gives an excellent description with photographs of a series of 20 cases in China,⁴¹ and later of 11 cases in New York,⁴⁶ of hæmolytic streptococcal gangrene affecting the skin and superficial tissues only, occurring chiefly on the limbs after a small injury or apparently spontaneously, the lesions frequently being multiple. The onset is acute, the patient prostrate, often with a positive blood culture. Hæmolytic streptococci are the only organisms invariably present. A better known subgroup of this variety, spontaneous hæmolytic streptococcal gangrene of the scrotum, has been recorded by several authors, Campbell's contribution⁴² with five of his own cases being a good example.

5 "Ecthyma gangrenosum" occurring in very young children in a poor state of health and frequently following a predisposing illness, usually varicella. The lesions are often multiple and may be extensive. Ecthyma gangrenosum may occur as a wound infection in debilitated children, especially where there has been contamination with urine or fæces. Lately several isolated cases, usually with multiple lesions, have been described in adults, and recently a more chronic form of pyoderma has been receiving a good deal of attention from German observers. A full list of references to these cases has been given by Brunsting,²² who described four cases of his own, one with extensive gangrene of the abdominal wall, in patients greatly debilitated with ulcerative colitis. Ecthyma gangrenosum is usually attributed to hæmolytic streptococci, and most authors regard it as a more extensive and deeper form of impetigo.

6 Infection with specific organisms. (a) Diphtheritic bacilli, as in the case of Smith,⁴³ where a spreading ulceration of the abdominal wall followed appendectomy, diphtheritic bacilli being isolated from the lesion, which responded well to anti-diphtheritic serum. (b) Blastomycotic and actinomycotic organisms. (c) Tubercle bacilli. (d) Leishman-Donovan bodies, as in oriental sores. (e) The lymphogranulomatous ultra-microscopic virus which causes intractable ulcers when the buboes break down.

7 "Amœbiasis cutis" may give a somewhat similar picture, as can be seen

by the descriptions of Heimbürger,¹⁶ who reviewed ten cases, most of which followed rupture of amœbic abscesses. He found that they responded promptly to emetine. Cole and Heideman¹⁷ describe a case of superficial gangrene following drainage of an appendix abscess with appearances identical with many in this series, in which amœbæ were consistently present in the pus exuded from the ulcerating edges, though there was no history of dysentery and examination of the appendix showed no amœbæ. The spread of the process was definitely checked by emetine hydrochloride intravenously, but there was relapse as soon as the drug was stopped. Eventually it was necessary to excise the margins, after which satisfactory healing promptly occurred. The features were so similar to other cases described in this series that they suggested that "progressive superficial gangrene" was due to infection with amœbæ. The failure of emetine to cure the condition and the final necessity of excision suggests that this particular case was not pure amœbiasis cutis. In no other cases have these organisms been discovered, and Brunsting²² and Meleney²³ and others, having read this suggestion, have had their cases examined by special experts, with negative results.

8 Foul sloughing infections may occur in wounds produced by human bites and where human saliva appears to have infected the deeper parts of the wound. The gangrene involves the deep tissues, and multiple sinuses are produced. The infecting organisms are fusiform bacilli and spirochætes. Flick described five cases and has reviewed the literature.²¹

ETIOLOGY

In none of the reported cases have any specific organisms been found except amœbæ in the case already discussed. In the majority of cases where full reports are given repeated special examinations have been made to exclude diphtheria, tuberculosis, blastomycosis, etc. In smears and cultures from the discharging pus a variety of organisms have been described. Streptococci were found in all 19 instances where bacteriological reports are included, 14 cases being non-hæmolytic. Staphylococci were found in 11 cases, diphtheroid and Gram-negative bacilli were frequently present.

In none of the cases was there evidence of any underlying disease. In no case was glycosuria reported, and the Wassermann reaction was always negative. In view of the not infrequent chronic ulcers found on the legs of patients with acholuric jaundice,³⁹ it is interesting to note that in our case three other members of the family were suffering from acholuric jaundice. Our patient, however, showed no evidence of the disease beyond a very slight increase in the fragility of the red cells. Lynn²⁴ was impressed by the fact that all the cases occurred in patients who required prolonged drainage and restricted diet and were consequently in a run-down general condition. He thought it significant that 50 per cent of the cases occurred after the age of 45. But many of the cases occurred in previously healthy people after an acute infection of only a few days, and a third of the cases occurred in the prime of life below 40. Lowering of the general resistance no doubt assists in the establishment of the infection, but it is not of fundamental importance.

There is no evidence to support as general etiological factors the suggestions of Borelli,³ who blamed the use of tincture of iodine in his case, and of Tennant,²⁰

who, having seen the two cases^{19, 20} in which cholecystitis had previously been present, thought they were due to hepatic insufficiency

With the observations of Arthus and others it is well established that frequent cutaneous necrosis due to hypersensitivity occurs in rabbits and guinea-pigs that have repeatedly been used for injections of bacterial and protein products. Necrosis of the skin at the point of injection has been observed clinically in human beings after diphtheria antitoxin and scarlet fever serum by Gatewood and Baldrige⁴⁷ and by others since. One dramatic case⁴⁶ is described of a girl who, having received seven weeks previously scarlet fever serum prophylactically, was given a large dose of the serum into the buttock four days after developing scarlet fever. A rapidly-spreading acute superficial gangrene followed over the whole of the buttock, thigh, and abdomen, which was cured by excision and skin-grafts. It was believed that the gangrene was due to the second injection being given while the patient was in a hypersensitive state. Ballin and Moore come to the conclusion that their cases^{27, 29} of post-operative progressive superficial gangrene are examples of the Arthus phenomenon. They then describe a series of inoculation experiments into the skin of rabbits of a mixed culture of streptococci and diphtheroid bacilli obtained from one of their cases. In one rabbit re-injection with a large dose after a sensitization period resulted in an ulcer with undermined edges and a central slough. Hypersensitivity may be a factor in the production of these post-operative cases, but until further proof is forthcoming it cannot yet be used to explain the whole process. It may be pointed out that Ballin and Moore had only one positive experiment, and none of the clinical cases following serum injections have completely resembled the slow progressive gangrene which is so characteristic.

The most important contribution towards the understanding of the etiology comes from Meleney,⁴ who in 1926 was able to grow from the excised lesion a micro-aerophilic streptococcus in cultures taken from regions outside the oedematous margin, and streptococci and staphylococci in cultures from the sloughs, the streptococcus appearing to prepare the way for the actual production of the gangrene by the staphylococcus. These organisms inoculated together into guinea-pig or rabbit's skin invariably produced a gangrenous ulcer, while either of them injected separately failed to produce gangrene. Later in 1931 he was able to confirm these findings on a second typical case²³. The same micro-aerophilic streptococcus was present in the periphery of the lesion, and again a lesion could only be produced by injecting both organisms together after mixing the cultures. Further, he showed that the association must be extremely close, as if injected separately, even in the closest juxtaposition or after an interval, no ulceration occurred. He found that the staphylococcus was not specific, as certain other staphylococci would produce a similar but not so marked lesion. He concluded that progressive gangrene is caused by synergistic activity of a symbiosis of a micro-aerophilic streptococcus and a staphylococcus. This streptococcus belongs to a group called by Prevot⁴⁹ *Streptococcus evolutus*, as they are anaerobic on first culture but after a number of plantations can grow aerobically. This organism is frequently found in the human intestine and in peritoneal exudates, and Prevot found them in the pus of all cases in a series of lung abscesses and bronchiectatic cavities. It is significant that 34 of the 37 cases of progressive gangrene have occurred after operations involving contamination with intestinal organisms or after empyemata. In 1933 Meleney had the opportunity of examining bacteriologically three further cases and was

again able to demonstrate the same streptococcus in each case. It is highly important that further bacteriological experiments on these lines should be carried out by those who may meet these rare cases in the future.

The infection has appeared to start round tension sutures in many of the cases and special emphasis has been placed on this by several authors,^{3, 13, 17, 23} and Patterson³⁰ even suggests that as a prophylactic measure tension sutures should never be used in cases of abdominal and chest abscesses. Tension sutures would therefore appear to be a factor in establishing the infection.

TREATMENT

A study of the results of treatment leaves no doubt as to the correct therapeutic procedure. Non-operative measures have all proved unavailing. These have included high vitamin diets, analgesics, and sedatives for sleep, all varieties of local applications, antiseptic and hypertonic, heat, X-rays, direct and artificial sunlight, which usually lead to irritation, metallic injections of arsenic, antimony, and manganese, antisera, vaccines, staphylococcal toxoid, protein shock, diphtheritic antitoxin, and repeated blood transfusions. In the 37 cases there appear to be only 3 exceptions, one after two months responded to quartz light,¹⁶ one to 6 per cent sodium chloride after involving the entire abdomen and both flanks,¹¹ and credit was given to general hygienic measures and the use of immunized blood transfusions in the third case, which had failed to respond to repeated attempts at excision.¹⁸ Of the others not treated surgically, 3 of the post-empyemata cases died after many months, the greater part of the trunk being involved,^{21, 22, 36a} one died of nephritis before excision could be carried out,²⁰ having refused excision, another recovered after ten months of pain and suffering when the lesion "burnt itself out",¹² the remaining one healed spontaneously after four months of such intolerable pain that the patient's morale was undermined and suicide was committed shortly afterwards.²⁰

All cases in which radical measures were adopted recovered with two exceptions, one a man of 70 who died of pneumonia²⁶ and another who died of endocarditis,³¹ the spread in both cases having been arrested. An incision with a cautery knife should be made beyond the advancing margin through the healthy skin and subcutaneous tissue down to the muscular layer so as to circumscribe the lesion. Excision of the entire ulcerating edge is then carried out. If a scalpel is used in extensive lesions as in two cases,^{1, 15} the tissues must then be coagulated in order to check the hæmorrhage, which may be so considerable as to render blood transfusions necessary.² If the lesion is extensive, the excision may have to be done in several stages, as in Christopher's case.² It was only necessary to cauterize the base in one case.¹⁵ The results were almost without exception immediate and gratifying, a pleasing feature being the instant cessation of the severe pain. Inadequate excision, however, was usually followed by prompt recurrence necessitating a repeat of the operation.^{4, 14, 18, 21, 30} Thiersch or pinch skin-grafts (used in 7 cases) may be applied after a short interval to assist healing, but the lesions usually heal spontaneously and sufficiently rapidly, especially where islands of new epithelium are present in the centre round the original wound. Should the gangrenous ulcer already be so far advanced that complete excision of the edges appears too formidable an operation, a barrier may be made in the healthy skin

beyond the œdematous margin by circumscribing the lesion with an incision down to the muscle and perhaps packing with 1 per cent formalin⁴ In all three cases^{1, 10, 32} where this procedure was adopted the spread was arrested and satisfactory healing resulted

It is hoped that in future this type of gangrene will be recognized during the first few weeks and the whole lesion immediately excised The scalpel should then be used for preference, as no time is lost while the tissue destroyed by the cautery is separating

SUMMARY

1 A case of a rare complication suitably called "progressive post-operative gangrene of the skin" is described The importance of using a definite nomenclature to facilitate recognition and reference is emphasized

2 Only one other case has been recorded in England, but 37 cases have been collected from the foreign literature and reviewed Their similarity justifies grouping them together as an entity

3 The rarity of the condition is shown by an analysis of all cases of post-operative gangrene of the abdominal and chest wall occurring in the London Hospital from 1903-33 One case only may be an example of "progressive post-operative gangrene of the skin", but the evidence available is too scanty to include it in the series

4 The most characteristic features are Its occurrence usually after drainage of an appendix abscess or empyema, the slow but steadily progressive spread, the involvement of superficial tissues only, the raised, œdematous, undermined and exquisitely tender edges, the relatively healthy base of red granulation tissue, the healing of the original sinus, the extensive area involved unless checked, and its failure to respond to any non-operative therapeutic measures

5 Treatment consists of early recognition and excision of the entire lesion, or if first seen in the later stages, of excision of the advancing edges with the cautery The results are immediate and gratifying

6 Bacteriological experiments suggest that it is due to the synergistic activity of a symbiosis of a micro-aerophilic streptococcus present in the pleural or peritoneal exudate and a non-specific staphylococcus introduced from without Tension sutures appear to play a part in the establishment of the infection Cutaneous hypersensitivity (similar to the Arthus phenomenon) may be an important etiological factor

7 The condition is little known in this country Failure to recognize it may lead to failure to carry out the only effective treatment, with consequent death of the patient or months or even years of pain and suffering

I wish to express my gratitude to Dr Hutchison and Dr Rowlands for their kind permission to publish this case

REFERENCES

Case References—

- ¹ CULLEN, T A, *Surg Gynecol and Obst*, 1924, xxviii, 579
- ² CHRISTOPHER, F, *Surg Clin N Amer*, 1924, iv, 795
- ³ BORELLI, C, *Giorn Ital d Mal Ven*, 1924, lxxv, 326

- ⁴ ⁵ ⁶ BREWER, G E, and MELENEY, F L, *Ann of Surg*, 1926, lxxiv, 438
⁷ ⁸ ⁹ PORTER, C A, *Trans Amer Surg Assoc*, 1926, xlv, 408
¹⁰ MOSCHOVITCH, *Ibid*
¹¹ CLINTON, *Ibid*
¹² ALEXANDER, E G, *Ann of Surg*, 1926, lxxiv, 461
¹³ MAYEDA, T, *Dent Zeits f Chir*, 1926, cxcix, 350
¹⁴ SHIPLEY, A N, *Ann of Surg*, 1928, lxxviii, 245
¹⁵ GILLESPIE, M G, *Ibid*, lxxviii, 248
¹⁶ GORDON, *U S Vet Bur Bull*, 1928, iv, 1045
¹⁷ COLE, W R, and HEIDEMAN, M L, *Jour Amer Med Assoc*, 1929, xcii, 537
¹⁸ PROBSTEN, J G, and SEELIG, M G, *Surg Gynecol and Obst*, 1928, lvi, 247
¹⁹ FREEMAN, L, *Ann of Surg*, 1930, xcii, 779
²⁰ TENNANT, *Ibid*, 783
²¹ POATE, R G, *Med Jour of Australia*, 1930, ii, 398
²² BRUNSTING, L A, *Arch Dermatol and Syph*, 1930, xii, 655
²³ MELENEY, F L, *Ann of Surg*, 1931, xciv, 961
²⁴ LYNN, F S, *Jour Amer Med Assoc*, 1931, xcvi, 1597
²⁵ WARFIELD, L M, *Ann Clin Med*, 1927, v, 884
²⁶ MITCHELL, J F, Read before Baltimore City Medical Society, 1930, Dec 5 Quoted by Lynn
²⁷ ²⁸ ²⁹ BALLIN, M, and MORSE, P F, *Amer Jour Surg*, 1931, vi, 81
³⁰ PATTERSON, A P, *Ann of Surg*, 1932, xcvi, 1091
³¹ BAKER, W H, and TERRY, C C, *Jour Amer Med Assoc*, 1932, xcvi, 138
³² SCOTSON, F H, *Lancet*, 1933, i, 80
³³ ³⁴ ³⁵ MELENEY, F L, *Surg Gynecol and Obst*, 1933, lvi, 847
³⁶ CAROL, W L, *Nederl Tijds v Geneesk*, 1932, lxxvi, 1838
^{36a} STEWART-WALLACE, A M Reported above

Other References—

- ³⁷ MELENEY, F L, *Ann of Surg*, 1930, xcii, 289
³⁸ McDONAGH, J, *Lancet*, 1933, i, 163
³⁹ VAUGHAN, J, *The Anaemias*, 1934 London
⁴⁰ HORSLEY, J S, *Arch of Surg*, 1929, vi, 882
⁴¹ MELENEY, F L, *Ibid*, 1924, ix, 317
⁴² CAMPBELL, M F, *Surg Gynecol and Obst*, 1922, xxxiv, 780
⁴³ SMITH, S W, *Brit Med Jour*, 1931, ii, 800
⁴⁴ STANNUS, H S, *Proc Roy Soc Med*, 1932, Nov
⁴⁵ HEIMBURGER, L F, *Arch Dermatol and Syph*, 1925, vi, 49
⁴⁶ MELENEY, F L, *Ann of Surg*, 1930, xcii, 289
⁴⁷ GATEWOOD and BALDRIDGE, *Jour Amer Med Assoc*, 1927, lxxviii, 1068
⁴⁸ MELENEY, F L, *Ibid*, 1929, xcii, 2009
⁴⁹ PREVOT, A R, *Ann de l'Inst Pasteur*, 1925, xxiv, 417
⁵⁰ MOORE WHITE, M, *Brit Med Jour*, 1934, ii, 932
⁵¹ FLICK, J B, *Ann of Surg*, 1932, xcvi, 118

ADENOMA OF THE DUODENUM

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ADENOMA OF THE DUODENUM

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EVERY writer on the subject emphasizes the comparative rarity of benign tumours of the gastro-intestinal tract. When the duodenum alone is considered the rarity is greatly enhanced. A review of the literature reveals the following important references —

Smoler¹ refers to an instance of adenoma of the duodenum recorded by Cruveilhier² in 1835. Rokitsansky,³ in 1861, when discussing connective-tissue tumours of the intestine, stated that non-malignant tumours of the duodenum may press upon the bile and pancreatic ducts causing a retention of their contents, but he gave no specific case reports. Salvioli,⁴ in 1876, reported an adenoma of Brunner's glands. Perry,⁵ in 1893, described a case of papilloma arising in the neighbourhood of the duodenal papilla and causing jaundice, microscopically it proved to be an adenoma composed of mucous glands. Thompson,⁶ in 1897, showed a specimen of polypoid growth of the duodenum at a meeting of the Pathological Society of Manchester, microscopically the tumour was a fibro-adenoma. Funkenstein,⁷ in 1904, reported multiple mucous-cell adenomata in the alimentary tract of a 24-year-old woman, six of which were in the duodenum. Doering,⁸ in 1907, noted an instance of diffuse polyposis of the intestines, several of these polypi being in the duodenum, and although the patient—a man—was only 28 years of age, the condition was associated with carcinoma of the transverse colon. The same author collected 50 cases of polyposis of the gastro-intestinal tract, but in 3 only was the duodenum involved. In 1908 Verse⁹ reported a case in which two adenomata were found in the duodenum. In 1910 Wechselmann¹⁰ referred to an instance of mucous-cell polyposis of the intestinal tract in which the duodenum was involved. In 1916 Weishaupt¹¹ described a duodenal tumour consisting of Brunner's glands with a fibromuscular stroma in a child 11 days old. Willis and Lasersohn,¹² in 1925, reported two instances of adenomata of the duodenum. In 1928 Golden¹³ added two cases to the list—one of adenoma of Brunner's glands and the other a mucous-cell adenoma which could be partially pushed back through the pylorus. This worker brought the total of non-malignant tumours of the duodenum up to 19, but this list included myoma, hæmangioma, papilloma, and others outside the adenomatous group. In 1929 Balfour and Henderson¹⁴ reported 6 cases of benign tumours of the duodenum, 3 of which were adenomata. They refer to the fact that in 119 cases of benign tumour of the intestine recorded by King, the duodenum was involved in only 5 instances, again illustrating the rarity of benign tumours in this situation. In 1933 La Roque and Shifflett¹⁵ reviewed the literature to date, but added little to the foregoing as far as benign tumours are concerned.

The description of the microscopic appearances of one of the adenomata reported in the literature (Willis and Lasersohn,¹² p 955) suggests that this tumour was really a fibroma covered by normal mucous membrane, and there may be a few others included in this group which are not true adenomata. It will therefore be realized that a Brunner adenoma is a rare pathological entity, and for this reason we present the following case.

CASE REPORT

G. B., a man aged 54 years, was admitted to hospital on April 23, 1934. He had had attacks of epigastric pain for the past nine months. The pain occurred about eight hours after food and was relieved by vomiting. During the week before admission he had vomited a considerable quantity of coffee-ground material and had had melena.



FIG. 445.—Adenoma of duodenum. Pyloric end of the stomach and first part of the duodenum.

Examination of the abdomen revealed tenderness on deep palpation over the epigastrium, but no tumour could be felt. A test-meal showed a rather high gastric acidity and a positive benzidine test on the fasting specimen, and a barium meal demonstrated a penetrating ulcer high up on the lesser curve of the stomach. The patient was examined at the request of Dr. A. V. Campbell, and it was decided that as the ulcer was a simple one medical treatment should be continued.

He was treated by diet, alkalis, and belladonna and discharged from hospital on June 11, with no symptoms and having gained 7 lb. in weight.

The symptoms, however, soon recurred and he was re-admitted on Sept. 1. A further X-ray was taken and showed the same ulcer and a duodenal cap which was normal in outline.

and filled well. Surgical treatment was then deemed advisable. However, at this time the patient's general condition was poor, as he had an obvious secondary anaemia and a bronchitis with some dullness over the right chest. The bronchitis improved with treatment and he was given a blood transfusion (16 oz.) two days before operation.

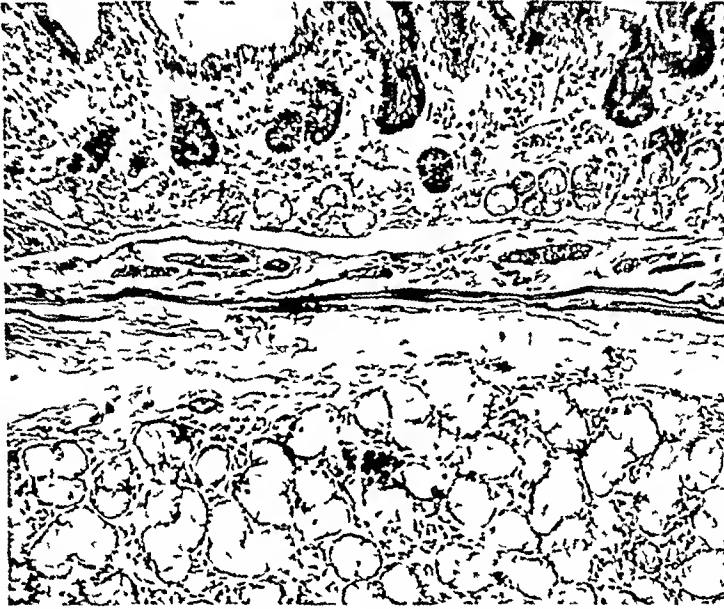


FIG 446—Adenoma of duodenum ($\times 140$)

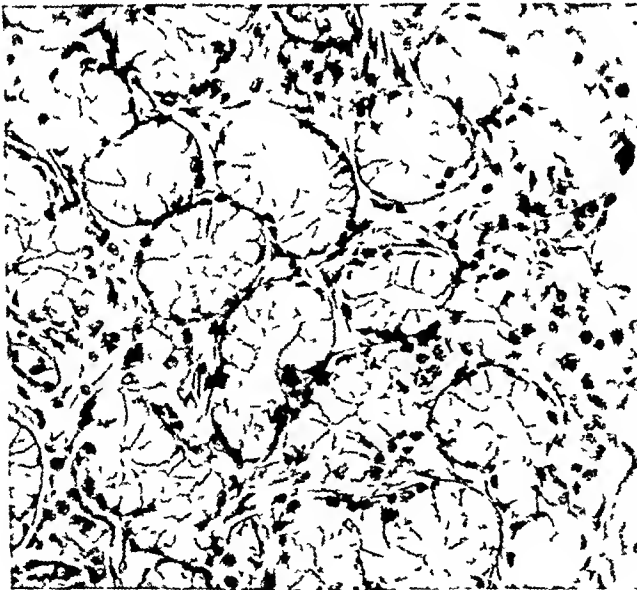


FIG 447—Adenoma of duodenum ($\times 400$)

OPERATION (Sept. 24).—The anaesthesia used was intratracheal gas and oxygen and local infiltration of the abdominal wall with 1 per cent novocain. The abdomen was opened through a right paramedian incision and the ulcer found rather high up on the lesser curvature.

A partial gastrectomy was decided on, and while palpating the pyloric end of the stomach a tumour was felt in the first part of the duodenum. This was about the size of a small plum and freely movable, obviously pedunculated. The presence of the tumour caused no difficulty in the operation, but rather more of the duodenum than usual was removed. A Billroth I type of anastomosis was performed, and the condition of the patient at the end of the operation was quite satisfactory.

SUBSEQUENT PROGRESS—Post-operative recovery was uneventful. The patient was allowed up on the twelfth day and was transferred to a convalescent home on the seventeenth day. His doctor reports that since he has returned home he has been quite well.

On reconsideration of this patient's history and examination in view of the operative findings, there does not appear to be anything more than can be fully accounted for by the gastric ulcer, and it seems probable that the duodenal tumour was not giving rise to any signs or symptoms.

PATHOLOGICAL REPORT—The specimen (Fig. 445) consists of the pyloric end of the stomach and the first part of the duodenum. There is a submucous hæmorrhage 1 in long just proximal to the pylorus. The duodenum contains a pedunculated mass, the pedicle being attached to the posterior duodenal wall about $\frac{1}{2}$ in distal to the pyloroduodenal junction. The pedicle gradually expands into a pear-shaped tumour of firm consistence and shows slight nodulation. There is no ulceration. Total length of polypus 1 $\frac{1}{2}$ in, circumference of pedicle at its narrowest part $\frac{1}{2}$ in, circumference of tumour at its widest part 3 $\frac{1}{2}$ in.

Microscopically (Figs. 446, 447) there is extensive hyperplasia of the duodenal glands, mainly beneath the muscularis mucosæ, and although these glands show some slight variation in size, their architectural features are well preserved and there are no abnormal mitotic figures. The tissues of the bowel are displaced to accommodate the mass, but there is no infiltration. The stroma is sparse and consists of loose connective tissue containing congested blood-vessels and there are scattered accumulations of small round cells. The mucous membrane covering the tumour is intact.

This microscopic picture closely resembles the description which Golden gave of one of his cases in 1928. He also found accumulations of round cells scattered throughout the tumour which were very similar to those described in the present instance. The condition is well shown in the photomicrographs.

DISCUSSION

Consideration of the recorded cases shows that many of the benign tumours of the duodenum do not cause any clinical symptoms. This is in direct contrast with similar tumours in other parts of the gut, which are so liable to form the starting-point of an intussusception. It is probable that the relative fixation of the duodenum prevents a similar occurrence in this part of the intestine.

While it is true that in one of the reported cases⁶ an adenoma growing from the region of the pylorus is stated to have caused partial invagination of that structure into the duodenum, the patient had no symptoms referable to this during life, and it can hardly be classed as an intussusception.

The earlier literature on the subject is chiefly made up of cases found at autopsy, or, since the more frequent resort to laparotomy, of those in which the duodenal tumour has been discovered during the course of an operation for some other disease. In fact, the number of cases in which a duodenal tumour has been the only lesion found, and so may be presumed to be the cause of the symptoms noted, is so small that a true symptomatology is still very difficult.

There does not appear to be any special age incidence, the youngest patient being 11 weeks old and the oldest 75 years. Intestinal obstruction is a rare event, and in the few cases in which it has been recorded the tumour has been very large and palpable through the abdominal wall. As a rule the growth is small, and

although some degree of dilatation may be present, the fluid contents of this part of the gut readily pass, even where the lumen is considerably diminished

When symptoms are present they are usually epigastric discomfort and perhaps pain of a colicky type, but seldom as severe as that associated with an ulcer. Nausea or vomiting is not uncommon and attacks of diarrhoea have been noted several times. The presence of melæna or occult blood in the stools would appear to be the most constant feature of significance and is the only one likely to be of diagnostic aid in the clinical investigation of the patient. The combination of melæna with epigastric symptoms is most likely to suggest the diagnosis of gastric or duodenal ulcer or gastric carcinoma, and the test-meal, and more especially the X-ray examination, are then the most important guides.

In recent years attention has been more keenly focused on the possibility of X-ray diagnosis, and emphasis has been laid on the advantages of fluoroscopic examination with changes of posture in addition to the routine radiogram, but even with up-to-date technique several cases have defied detection. When diagnosis by Roentgen ray is successful there is usually a filling defect of vacuolation type. Golden attaches considerable importance to the presence or absence of a six-hour gastric retention as a means of distinguishing growths arising in the duodenum from those prolapsing into it from the stomach. He states that "in the presence of a filling defect in the duodenal bulb suggesting non-malignant tumour, a six-hour gastric retention may be considered as evidence in favour of a growth arising in the stomach and prolapsing into the duodenum, and the absence of such a retention is indicative of a growth arising in the duodenum itself". Waters¹⁶ points out that a vacuolated type of filling defect points to adenoma, whereas a multilocular filling defect indicates papilloma.

A considerable proportion of instances of benign tumours of the duodenum is associated with pathological conditions elsewhere in the gastro-intestinal tract, such as multiple polyposis, carcinoma, cholelithiasis, and ulceration. The relation of ulceration to lesions in the duodenal glands deserves careful consideration in view of the recent work by Florey and Harding,¹⁷ whose observations give additional support to the view that the normal secretions of Brunner's glands protect the mucosa from damage by the acid gastric juice by virtue of their mucoid consistence and contained alkali. These observers are of opinion that their findings lend further weight to the suggestion that malfunctioning of these glands may be primarily responsible for duodenal ulcer.

As regards treatment, since the presence of a duodenal tumour is so often only discovered during an operation for some other condition, the surgeon must often be guided by circumstances as to the best treatment to adopt. When the associated lesion is a gastric or duodenal ulcer, a radical removal of the ulcer and the tumour would seem the correct method. There is, however, no recorded case of a simple duodenal tumour becoming malignant, and where the growth is polypoid trans-duodenal resection should suffice. In the case of a sessile growth or one in which the possibility of sarcoma or carcinoma cannot be definitely excluded, the portion of duodenum involved should be excised and an end-to-end anastomosis performed.

Our thanks are due to Dr T V Cooper for supplying some of the references, to Dr Campbell for his notes on the case, and to Mr W H Fussell for his help in preparing the photomicrographs.

REFERENCES

- ¹ SMOLER, F, *Beitr z klin Chir*, 1902, *xxvi*, 139
- ² CRUVEILHIER, J, *Anatomie path du Corps humain*, 1835-42, *xii*, 6, from Smoler
- ³ ROKITSANSKY, K, *Lehrbuch der pathologischen Anatomie*, 1861, 3rd ed, 231 Vienna W Braunmuller
- ⁴ SALVIOLI, G, *L'Osservatore, Gaz del clin di Torino*, 1876, *xii*, 481
- ⁵ PERRY, E C, *Trans Pathol Soc Lond*, 1892-3, *xiv*, 84
- ⁶ THOMPSON, P, *Lanet*, 1897, *i*, 383
- ⁷ FUNKENSTEIN, O, *Zeits f klin Med*, 1904, *lv*, 536
- ⁸ DOERING, H, *Arch f klin Chir*, 1907, *lxxviii*, 194
- ⁹ VEPSE, M, *Arb a d path Inst in Leipzig*, 1908, *v*, 1
- ¹⁰ WECHSELMANN, L, *Beitr z klin Chir*, 1910, *lxx*, 855
- ¹¹ WEISHAUP, ELISABETH, *Virchow's Arch*, 1917, *cciii*, 24
- ¹² WILLIS, A M, and LASERSON, M, *Ann of Surg*, 1925, *lxxii*, 952
- ¹³ GOLDEN, R, *Amer Jour Roentgenol*, 1928, *xx*, Nov, 405
- ¹⁴ BALFOUR, D C, and HENDERSON, E F, *Ann of Surg*, 1929, *lxxix*, Jan, 30
- ¹⁵ LA ROQUE, G P, and SHIFLETT, E L, *Ibid*, 1933, *xcviii*, Aug, 178
- ¹⁶ WATERS, C A, *Amer Jour Roentgenol*, 1930, *xxiv*, Nov, 554
- ¹⁷ FLOREY, H W, and HARDING, H E, *Jour Pathol and Bacteriol*, 1934, *xxxi*, Sept, 255

MEASURED DOSAGE IN THE RADIUM TREATMENT OF CARCINOMA OF THE URINARY BLADDER

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OF late years much has been written on the radium treatment of carcinoma of the urinary bladder, and were the object of this contribution merely to state a case for the radiosensitivity of this particular neoplasm it might well be considered superfluous. But radium therapy is entering at the present time into a new phase, in which the old empiricism is being discarded in favour of a system of physically determined dosage, and this paper will show how such a system can be applied in practice to carcinoma of the bladder in such a way that the surgeon will know, firstly, that his dosage is safe, and secondly, that his dosage is the same for all tumours treated, irrespective of their size.

INTRODUCTION

No single dosage unit for gamma-ray therapy has so far found general acceptance, but the fact that a variety of such units is being pressed upon the radiological world from widely different sources goes to prove how general is the dissatisfaction with older methods. The particular unit adopted in any country or clinic matters little, so long as it is a genuine measure of the amount of energy delivered to the tumour and its bed in any stated case. Of these units, however, the 'roentgen' ('r' unit—gamma radiation) is the one which is probably most widely adopted at the moment and which is likeliest to gain international recognition, and it is here accepted.

Research has been active also along another line—the distribution of radioactive foci in any plan of treatment. The basic requirement is the production of a homogeneous field of irradiation which will include the tumour and its bed. If this can be achieved, elimination is secured, firstly of localized necrosis due to high dosage, and secondly of localized areas of sublethal dosage from which subsequent recrudescence is bound to occur in an otherwise adequately treated zone. It is apparent that two separate but interdependent factors are involved, namely (1) The total amount of radium required, (2) The number and distribution of the foci of radiation. These two factors are fully discussed in a recent article by Paterson and Parker on the application of "measured dosage" to the problems of radium therapy. The present paper deals with the application of the system there outlined to the special case of carcinoma of the urinary bladder. As will be seen in the following pages, the manner of the application is such as to be quite within the reach even of those who have no bent for considerations of pure physics.

TECHNIQUE OF TREATMENT

Preliminary investigations completed, suprapubic cystotomy is performed, and the bladder explored. The growth having been located, the best possible exposure is obtained. Often the bulk of a tumour, especially if pedunculated, prevents adequate access to its base and the surrounding tissues. In such cases it is recommended that the protuberant and obstructive portions be removed by endothermy merely down to the normal level of the mucosa, and without any attempt at complete treatment in this way. The full extent of the growth having been investigated, it remains now to radiate in the best manner possible.

The difficulties inherent in any method of surface application of radium within the bladder are so great that it cannot be said that any such technique based on scientific principles has yet proceeded beyond the purely experimental stage. For the present, therefore, it is desirable to consider only the interstitial approach to the problem.

The first step is to determine the area to be radiated, since the distribution of radio-active foci depends upon the shape and size of this area. Either a circle or a square may be chosen. As the former, however, is almost universally applicable in the bladder, and as the rules for circular implantations can be reduced to simple tabular form (as is shown later), attention will be confined to this distribution.

From the Appendix it will be seen that implantation of a circular zone demands, according to the size of the circle, one of three distributions: (1) An outer circle only, (2) An outer circle with centre point, (3) An outer circle with inner circle of half the radius of the outer one, and a central point. Now, it must be emphasized that the more numerous the foci the more satisfactorily can such distributions be achieved. If for no other reason, therefore, radon seeds would be the vehicles of choice, as being much smaller than any radium needles commonly in use. Their selection, however, is further indicated because, being more easily inserted than needles even when exposure is not of the best, their use shortens the operative procedure. Moreover, arrangements for subsequent removal are eliminated. Study of the Appendix will show how the required distributions of the seeds should be planned ideally, but the surgeon must find the most practicable way of applying these principles, and a rule-of-thumb method of doing so is set forth in *Tables I-III*. Each table is constructed in the same manner, and shows at a glance just how many seeds are required for a circle of any given size between diameters of 3 and 7 cm, and just how the physical requirements are most fully met. *Table I* gives instructions when seeds of 1.1 mc initial strength are used, this strength being considered best. *Tables II* and *III* apply to seeds of initial strength equivalent to 1.3 mc and 1.5 mc respectively, the latter being considered the maximum strength which can be used with any degree of safety. Such seeds, of filtration equivalent to 0.5 mm gold, have been found satisfactory in practice.

The seeds are implanted at a depth of $\frac{1}{2}$ cm below the mucosal surface, and the distributions given in the tables are such as to produce a dose of 6000 'r' units on each surface of a disc of bladder wall corresponding to the area treated and of a thickness of 1 cm. Some modification of this dosage level may be found desirable in the light of further experience, but present indications are that it is sufficient, without being dangerous to such healthy tissues as may lie within the disc in question. Every case should be implanted wide of the tumour in every

direction, a circle being selected of such size as to include the whole growth with a margin of healthy tissues around it. Accurate implantation is insisted upon, within the limits of practicability, and the number of seeds required for any circle is disposed at equal intervals around the circumference.

Table I—DISTRIBUTION OF RADON SEEDS OF INITIAL STRENGTH 11 mc

	DIAMETER IN CENTIMETRES OF THE VARIOUS CIRCULAR AREAS				
	3	4	5	6	7
Total seeds required	8	11	14	19	24
Number in outer circle	7	9	10	13	16
Number in inner circle	0	0	4	5	7
Centre point	1	2	0	1	1

Table II—DISTRIBUTION OF RADON SEEDS OF INITIAL STRENGTH 13 mc

	DIAMETER IN CENTIMETRES OF THE VARIOUS CIRCULAR AREAS				
	3	4	5	6	7
Total seeds required	7	9	12	16	21
Number in outer circle	6	7	8	11	14
Number in inner circle	0	0	4	5	6
Centre point	1	2	0	0	1

Table III—DISTRIBUTION OF RADON SEEDS OF INITIAL STRENGTH 15 mc

	DIAMETER IN CENTIMETRES OF THE VARIOUS CIRCULAR AREAS				
	3	4	5	6	7
Total seeds required	6	8	11	14	18
Number in outer circle	5	6	8	10	12
Number in inner circle	0	0	3	4	5
Centre point	1	2	0	0	1

A brief example is appended in order to demonstrate the procedure. Suppose we have a lesion of diameter 4 cm, such that a circle of 7 cm diameter be found adequate, and that seeds of 11 mc are available. It will be seen from Table I that twenty-four seeds will be required. 16 of these should be distributed equally around the circumference of a circle of 7 cm diameter, 7 around the circumference of a circle of 3.5 cm diameter, and 1 should be placed at the centre point. All should be approximately $\frac{1}{2}$ cm deep to the mucosa.

If seeds are not available, recourse must be had to radium needles. These have undoubted disadvantages. They take much longer to insert, and require subsequent removal. In addition, there is a greater liability to accidental injury by penetration of the bladder wall at the time of implantation. Scientific dosage according to modern standards is quite possible of achievement, but obviously the circular method is hardly possible. Some form of square implantation is indicated. The calculation is not difficult, but cannot be reduced to simple tabular form. Full details are available in the article by Paterson and Parker previously referred to.



FIG 448—Flexible rule (Dr Herd) (Manufactured by Messrs C F Thickrat, Leeds)

The taking of measurements within the bladder is not easy, but is facilitated by the ingenious flexible rule devised by Dr Herd, and shown in *Fig 448*. *Fig 449* shows a useful seed-introducer with handle set at right angles to the barrel. This arrangement obviates obstruction of the view by the hand. The point of the introducer is smeared with sterile vaseline, and the instrument is loaded from the distal end, the vaseline causing the seed to remain in the barrel even in the dependent

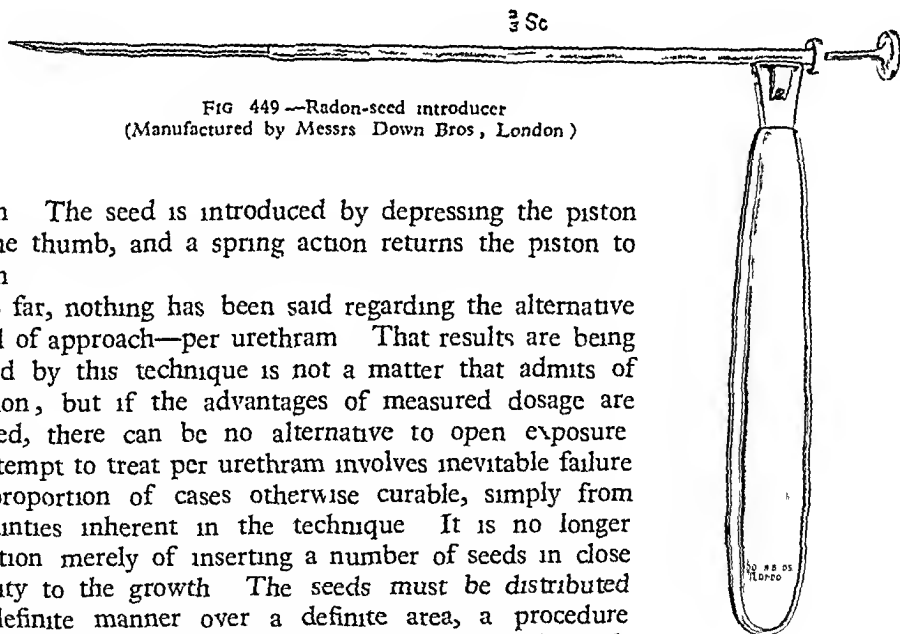


FIG 449—Radon-seed introducer
(Manufactured by Messrs Down Bros, London)

position. The seed is introduced by depressing the piston with the thumb, and a spring action returns the piston to position.

So far, nothing has been said regarding the alternative method of approach—per urethram. That results are being obtained by this technique is not a matter that admits of refutation, but if the advantages of measured dosage are conceded, there can be no alternative to open exposure. Any attempt to treat per urethram involves inevitable failure in a proportion of cases otherwise curable, simply from uncertainties inherent in the technique. It is no longer a question merely of inserting a number of seeds in close proximity to the growth. The seeds must be distributed in a definite manner over a definite area, a procedure sufficiently difficult when suprapubic exposure is obtained, and frankly impossible through the operating cystoscope. In short, radium implantation by the per-urethral route is a procedure which must be universally condemned when cancer is suspected. In support of this, the writer would quote Barringer in the statement that "even the smallest bladder tumour, if it be cancerous, is a grave disease not to be treated by inadequate means."

RESULTS OF RADIUM TREATMENT

In conclusion it must be emphasized that the omission of the consideration of purely surgical methods of dealing with carcinoma of the bladder is not an indication that these are regarded as being unimportant. Time alone will show which cases are best handled surgically and which best dealt with by radium. *Table IV* shows the results of treatment in the Radium Institute, Manchester, for 1931.

Table IV—RESULTS OF TREATMENT OF CARCINOMA OF THE URINARY BLADDER
(RADIUM INSTITUTE, MANCHESTER, 1931*)

Total cases treated			9
Alive	Well	3	} 4 (44.4 per cent)
	Diseased	1	
Dead	Post-operative	1	} 5 (55.5 per cent)
	Malignancy	4	

* Interval since treatment, over two and under three years

This represents the results as achieved in the early days of the present phase when knowledge of radium distribution was much less perfectly worked out than now, and when only inoperable cases were treated. Complete homogeneity of radiation could not be claimed, and yet the figures at a period of over two and under three years show 33.3 per cent of cases alive and well. As indicative of the type of case going to make up this group it may be stated that the smallest growth measured 4×3 cm (in this case a second smaller growth was present in addition), and that the remainder varied from this up to an enormous one measuring 14×12 cm. All except one involved the base of the bladder. In such cases most surgeons will prefer radium to extremely radical surgical extirpations with their high mortality and other attendant disadvantages. Differences of opinion must arise, however, in the early cases, and then the best interest of the patient can be served only by due consideration of the individual circumstances of the case by one who is fully alive to the possibilities of both radium and surgery.

SUMMARY

- 1 Recent advances in radium technique are described in their application to the treatment of carcinoma of the urinary bladder
- 2 A concise guide is supplied to the arrangements and amounts required in actual practice, based on the finding that 6000 'r' is the satisfactory dose
- 3 The question of the surgical approach is discussed, and cystoscopic methods of treatment are condemned
- 4 Some results are quoted

APPENDIX

By H. M. PARKER

PHYSICIST, RADIUM INSTITUTE, MANCHESTER

In planning a scheme of radium distribution to produce homogeneous and calculable radiation the use of circular arrangements is especially favourable. On the one hand, the circles have a wide range of clinical applicability, and, on the other hand, the difficulties of calculation are reduced to a minimum. The variation

of intensity over a plane at 0.5 cm from radio-active circles of different diameters is shown in *Fig 450*. It will be seen that the small circles give uniform irradiation, whereas the larger circles show a progressive weakening of the central intensities as the size increases. It is obvious from the shape of the distribution curves

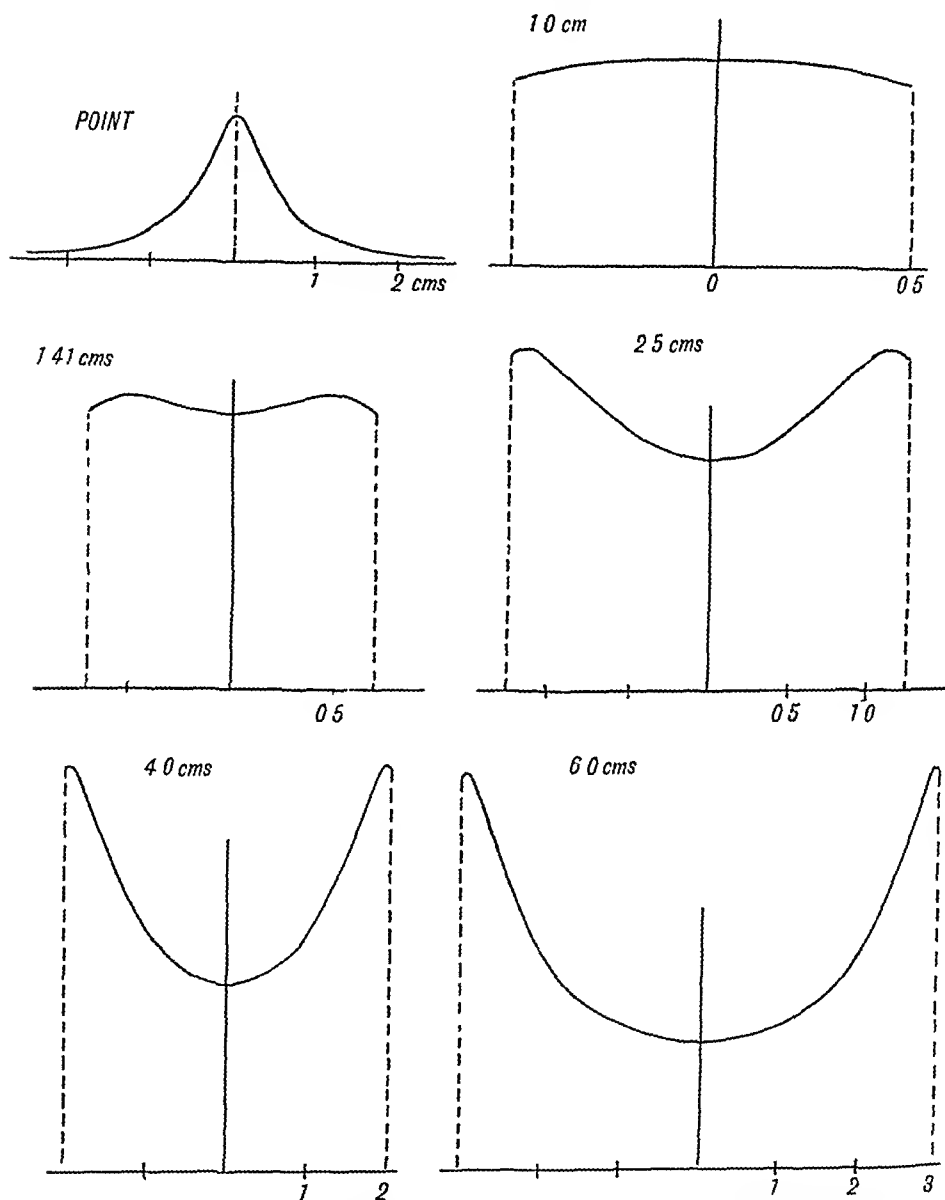


FIG 450—Distribution of intensity at 0.5 cm from radio active circles of different diameters. The dotted ordinates define the limits of the treated area

that for any total diameter it is possible to develop a combination of rings to deliver radiation homogeneous to any required degree. With a permissible variation of ± 10 per cent, a combination system possessing the necessary clinical simplicity

has been developed by R Paterson and the writer (*Brit Jour Radiol*, 1934, Oct) For diameters between 1.5 and 3 cm it is sufficient to place 5 per cent of the total radium at the centre For diameters of 3 or more cm, a centre-spot of 3 per cent and an inner circle of half the total diameter, with a particular percentage of radium, are adequate The modified distributions of intensity are shown in *Fig 451* The

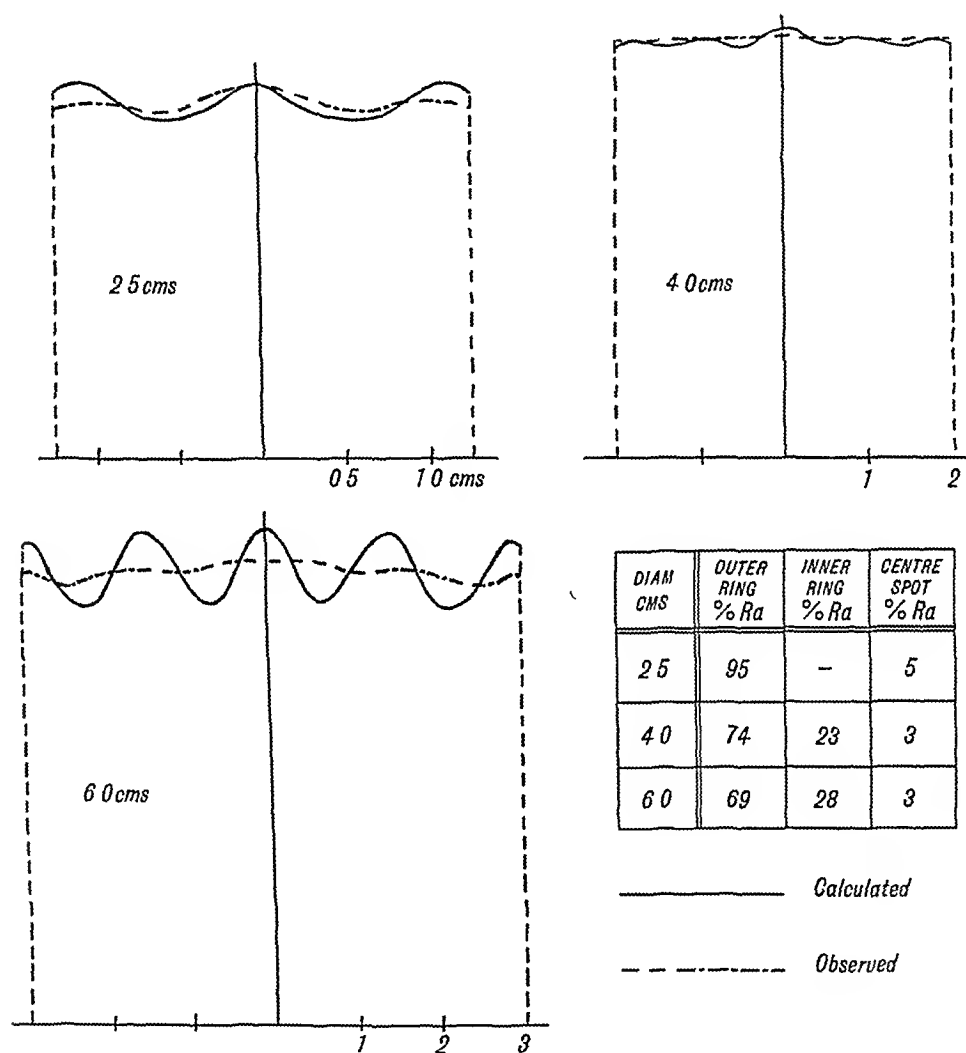


FIG 451—Calculated and observed distributions of intensity at 1 cm from three compound circular applicators The greater degree of homogeneity of the experimental curves is mainly due to the finite size of the micro ionization chambers The dotted ordinates define the limits of the treated area

required percentages can be readily assessed from the accompanying graph (*Fig 452*)

The arrangements outlined above were developed for superficial application, but they may be applied to interstitial treatment without alteration There will be two parallel planes 1 cm apart at which the dosage will be uniform and adjusted to say 6000 'r' Then no point between these planes will receive less than 6000 'r', but on the other hand the dosage within the disc will not greatly exceed 6000 'r'

except at local foci round each active source, a departure from homogeneity which is well known to produce no deleterious effect

Experiments with micro-ionization chambers show that the distribution and dosage in tissue agree very closely with those calculated in air on a simple basis

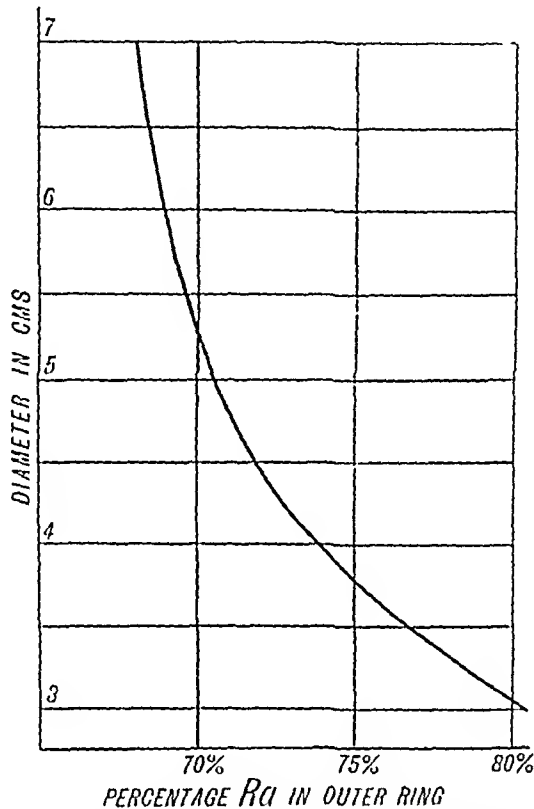


FIG 452—Graph showing the percentage of the total radium in the outer circle of the compound applicators 3 per cent is to be placed at the centre, and the remainder in the inner ring

Owing to the special type of gamma ray scattering there is no correction factor equivalent to 'back-scatter' in X-ray therapy

BIBLIOGRAPHY

- BARRINGER, B S, *Surg Gynecol and Obst*, 1934, lviii, May, 867
 PATERSON, R, and PARKER, H M, *Brit Jour Radiol*, 1934, vii, Oct, 592
 SMITH, A J DURDEN, *Brit Med Jour*, 1934, Sept 29, 584
 SOUTTAR, H S, *Radium and Cancer A Monograph*, 1934 London William Heinemann
 STEVENS, J THOMPSON, *Radiology*, 1934, xxii, Jan, 99

INTERINNOMINO-ABDOMINAL (HIND-QUARTER) AMPUTATION

By G GORDON-TAYLOR

SURGEON, MIDDLESEX HOSPITAL, LONDON

AND PHILIP WILES

ASSISTANT SURGEON, QUEEN'S HOSPITAL FOR CHILDREN, LONDON

INTERINNOMINO-ABDOMINAL amputation is fortunately an infrequent operation of surgery, and must remain for all time one of the most colossal mutilations practised on the human frame. Though this mighty ablation was first performed by Billroth¹ in 1891, to Girard² belongs the credit of having achieved, in 1895, the first success.

The term 'interinnomino-abdominal amputation' would seem to be the best and most appropriate appellation for this operation, for 'interpelvi-abdominal amputation', the title used by Hogarth Pringle,³ is rather suggestive of some feat of expert swordsmanship, and would appear to betoken an even more stupendous undertaking than that which is on occasion essayed by surgeons. It is true that in one of our cases a testicle required removal as well, that in another one line of bone section involved the ischial and pubic portions of the opposite innominate bone, and that in one successful case part of the sacrum required removal with the mass, nevertheless, this hyperbolic and ambitious nomenclature hardly seems justified, even although the usual limits of the amputation are transcended by these additional sacrifices of bone or gonad. 'Interilio-abdominal removal' might be applicable to certain more limited growths, but in the cases that have been encountered by the writers nothing but the sacrifice of the whole innominate bone seemed practicable, if the operation were to be effective in its object and purpose. 'Hemipelvectomy' evokes a shudder, and a sigh of regret that some knowledge of Latin and Greek is no longer considered necessary for those who claim to have received more than a kindergarten education.

The literature of the subject has been tabulated by Hogarth Pringle³ in this JOURNAL in 1916, and by Judin⁴ in 1926, the latter author, though writing in English, had been working in Russia, and, perhaps on account of the inaccessibility of references, his table contains a number of inaccuracies. Many of the cases reported in Russian papers and journals are unobtainable in London, and others were personal communications to Judin, so that an accurate scrutiny of these cannot be made. The perusal of the relevant surgical literature indicates, however, that the operation has now been performed at least 79 times by forty-eight different surgeons, but of the reported cases only those of Hogarth Pringle and the present series emanate from British clinics.

The opportunity presented to one of us (G G-T) of performing this amputation as a *single-stage operation* on no fewer than five occasions would seem to be unique. It is true that Hogarth Pringle in his memorable paper already mentioned reported five cases, but in three of these the operation was performed in two stages,

the time intervening between the two scances varying between three months and fifteen months, and in two of the cases the second operation was merely the ablation of a disintegrated os coxæ

Fortune has kindly ordained that the two authors of this communication have been associated together in the performance of the last four of the five operations that constitute the basis of this paper, and it is perhaps significant that of these four cases only one succumbed. Moreover, the last three cases of the series were operated upon within a few months of each other, and an unparalleled opportunity has thus been afforded us of not only familiarizing ourselves more thoroughly with the technique of the manœuvre, but also of reviewing the anxieties of the undertaking, its technical difficulties and pitfalls, and the post-operative complications. We are furthermore enabled to form some opinion as to the degree of final disablement produced by the ablation and to adjudge the amount of interference with the patient's subsequent *modus vivendi*.

Hogarth Pringle's most admirable paper has been of the utmost assistance to us in the operative treatment of the cases dealt with in this communication, and that author is indeed to be congratulated on his low mortality, when it is remembered that at the time his operations were performed blood transfusion was not readily available, at any rate in Great Britain, and the shock of the operation had to be combated with nothing more serviceable than saline infusion.

The conviction has grown upon us that some description of the operation as we have experienced it might be of value to those who may from time to time find themselves constrained to advise and to perform this amputation, and the sketches illustrating certain of the stages of the undertaking and the anatomical structures displayed in the course of its performance may be an aid to such surgeons as have less experience of this amputation than ourselves.

It is probable that in the future this operation will be less and less frequently performed. Radiation therapy at the moment seems to be displacing the more crude and more maiming methods of ablating cancer, but even with the growing perfection of methods of radiation, the treatment of certain forms of sarcoma may perhaps still occasionally demand this operation, and so long as procrastination remains a characteristic human trait where disease is concerned, and while benign or quasi-benign tumours are allowed to attain colossal and crippling proportions, only this type of procedure will rid the patient of his encumbrance, his pain, and his menace of malignancy.

CASE REPORTS

Case 1—Sarcoma of upper end of femur with involvement of innominate bone. Interinnomino-abdominal amputation. Death.

HISTORY—A H, male, aged 25. The patient's father had died of a sarcoma of the leg twenty-three years previously. The patient had complained of pain in the right hip for four years, but there had been no swelling during that period. He sustained a fall on May 16, 1922, after which he became lame. Radiography now revealed the true nature of the malady, and the report from another institution, dated May 17, 1922, was as follows: "Large periosteal sarcoma of the upper end of the right femur, extending from the region of the great trochanter on to the inferior surface of the neck of the bone and for six or seven inches down the shaft. There are also opacities in the acetabulum and ilium above, which may be secondary deposits. There is considerable outward bowing of the shaft of the femur." An X-ray report dated May 22, 1922—a week later—revealed a transverse fracture of the shaft at the lower end of the growth, which had increased in size in that short space of time. The patient was

transferred to my care (G G-T) at Middlesex Hospital as an inoperable case in the middle of June, 1922, and by this time there was radiological evidence of the involvement of the acetabulum by the neoplasm

ON EXAMINATION—The general condition of the patient was poor he was anæmic, his pulse was 100, and at times irregular, and he suffered untold pain in the region of the right hip and thigh. In the upper and middle thirds of the latter there was a large diffuse swelling, there was marked outward bowing. The right leg and foot were greatly swollen and there was considerable œdema. In the right groin was a hard, fixed mass, extending on to the abdominal wall. The circumference at the upper end of the thigh measured 56 cm on the right side and only 41 cm on the left. A diagrammatic sketch of the swelling is appended (Fig 453), the X-rays have unfortunately been destroyed.

It was resolved, if the peritoneum were free of involvement by the new growth, to attempt to eradicate the mass by an interinnomino-abdominal amputation.

OPERATION (July 7, 1922)—Spinal anæsthesia and general anæsthesia were administered by Mr A E W Idris. The steps of the operation were those outlined by Hogarth Pringle, but in this case the extent of the growth necessitated the removal of the right testicle as well. Very considerable hæmorrhage was encountered in the extraperitoneal tissues in the neighbourhood of the cut symphysis and the bladder, and despite the fact that the common iliac vessels had been already secured, much blood was lost. Unfortunately all the arrangements for blood transfusion miscarried, the arrangements for transfusion were not organized as they are now, nor were the services of the Blood-Transfusion Society available then. In our emergency we could only avail ourselves of the services of a diminutive "girl-guide", who donated with great spirit, but with little effect! The patient died of shock about six hours after the completion of the operation.

HISTOLOGICAL REPORT (No 4197/22, dated 26/7/22) —

Macroscopically the tumour had destroyed probably the whole thickness of femur for a distance of about three inches immediately below the trochanter. It had invaded the glands along the external iliac vessels, all except the most recently formed portions contained bone.

Microscopically the section showed the structure of a round-celled sarcoma. The cells showed a tendency to line the small alveoli formed by trabeculae of fibrous tissue.

AUTOPSY—There was one note of encouragement from the autopsy despite the magnitude of the primary growth there was a complete absence of any metastases throughout the body.

Case 2—Osteoclastoma of innominate bone Interinnomino-abdominal amputation Recovery

HISTORY—W J, male, aged 17, while playing football sustained a blow in Scarpa's triangle on the left side, the injury was sufficiently severe to lame him and he was unable to continue the game. His incapacity persisted and he consulted his doctor, a radiogram at this period revealed no bony change. For several weeks thereafter the patient was lost sight of by his medical attendant, having betaken himself to some unqualified persons who enjoyed the confidence and patronage of the chief football club of the town in which he dwelt. By these he was massaged and manipulated, but a swelling having now appeared in the adductor region of the left thigh in the vicinity of the cruro-scrotal fold, the patient returned to the doctor whom he had neglected and was promptly sent up to Middlesex Hospital.

ON EXAMINATION—A well-marked tumour could now be felt in the upper part of the thigh and in the perineum, where it could be identified on rectal examination and on deep palpation in the urogenital triangle and left ischio-rectal fossa, it was fixed to the pubic bone, was tender on pressure, and was much more considerable on the deep surface of the innominate bone than on its superficial aspect. All movements of the left hip were slightly limited and there was definite wasting of the quadriceps extensor muscle of the affected side.

Radiological Report (Middlesex Hospital)—The left ischio-pubic junction has almost

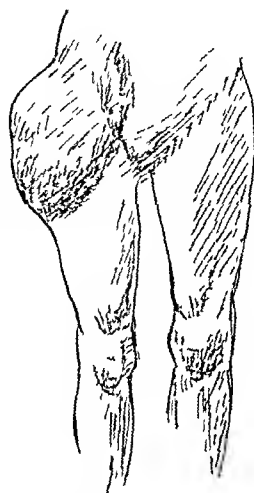


FIG 453 — Case 1
Diagram indicating the extent of growth. The upward extension of the growth on to the lower part of the abdominal wall is shown.

completely disappeared. There is irregular new bone formation in this region. The remainder of ischium is increased in density and there is a tumour with bone formation encircling it. Appearances point to osteosarcoma (*Fig 454*).

The clinical characters of the tumour, the rapidity of growth, the radiological report, and particularly the marked contrast between the first and second radiograms taken at a short interval of time, together with the history of injury, prompted the diagnosis of a sarcoma of the innominate bone, which was shared by several of my colleagues. The case was considered to be one more likely to be permanently benefited by amputation than by radium therapy, it was thought that the relative inaccessibility of the tumour might tend to imperfect radiation.



FIG 454—Case 2 Radiogram of cystic osteoclastoma of left innominate bone. Interilio abdominal amputation successfully performed, February, 1929.

OPERATION (February 4, 1929)—Interinnomino-abdominal amputation performed by G Gordon-Taylor. Spinal anaesthesia was induced by Dr H P Crampton, with heavy stovaine, this was supplemented by light open ether. Messrs Eric Riches, P Wiles, and H K Evans rendered loyal and valuable assistance.

The incision and the steps of the operation were mainly those outlined by Hogarth Pringle. It was deemed important to satisfy oneself at the earliest moment as to the relations of the tumour to the peritoneum, and to convince oneself of the possibility of ablating the neoplasm. The flat muscles of the abdominal wall were therefore cut through at their attachment to the iliac crest at the very commencement of the undertaking. The symphysis required division by sawing. In this case the common iliac vessels were not ligated, the external iliac artery and vein were tied distal to the origin of the deep epigastric vessels. The nerves were injected with novocain and were treated with 80 per cent alcohol after section. The iliac portion of the innominate bone was sawn through lateral to its sacral articulation.

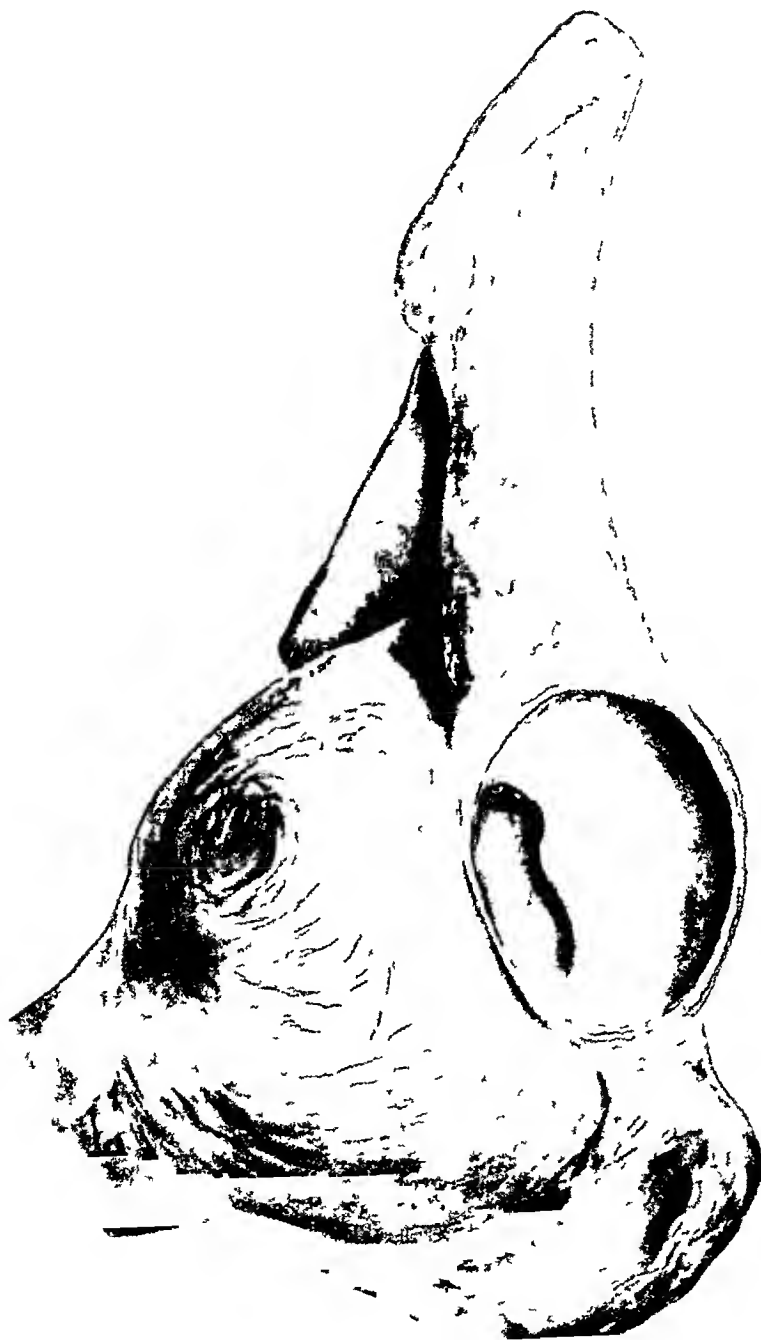


FIG 455—*Case 2* Naked-eye appearance of innominate bone, the seat of a cystic osteoclastoma
Interilio-abdominal amputation successfully performed, February, 1929



FIG 456—Case 2 The same bone as Fig 455, showing the growth View from inner aspect

Subsequently this articular portion of the innominate bone was found to project unduly, and was easily and expeditiously removed. Immediately before operation 800 c c of blood were obtained from the patient's brother, citrated, and kept warm. This was given slowly, commencing immediately the main vessels were divided. A further transfusion of 400 c c from another donor was given some hours after the operation.

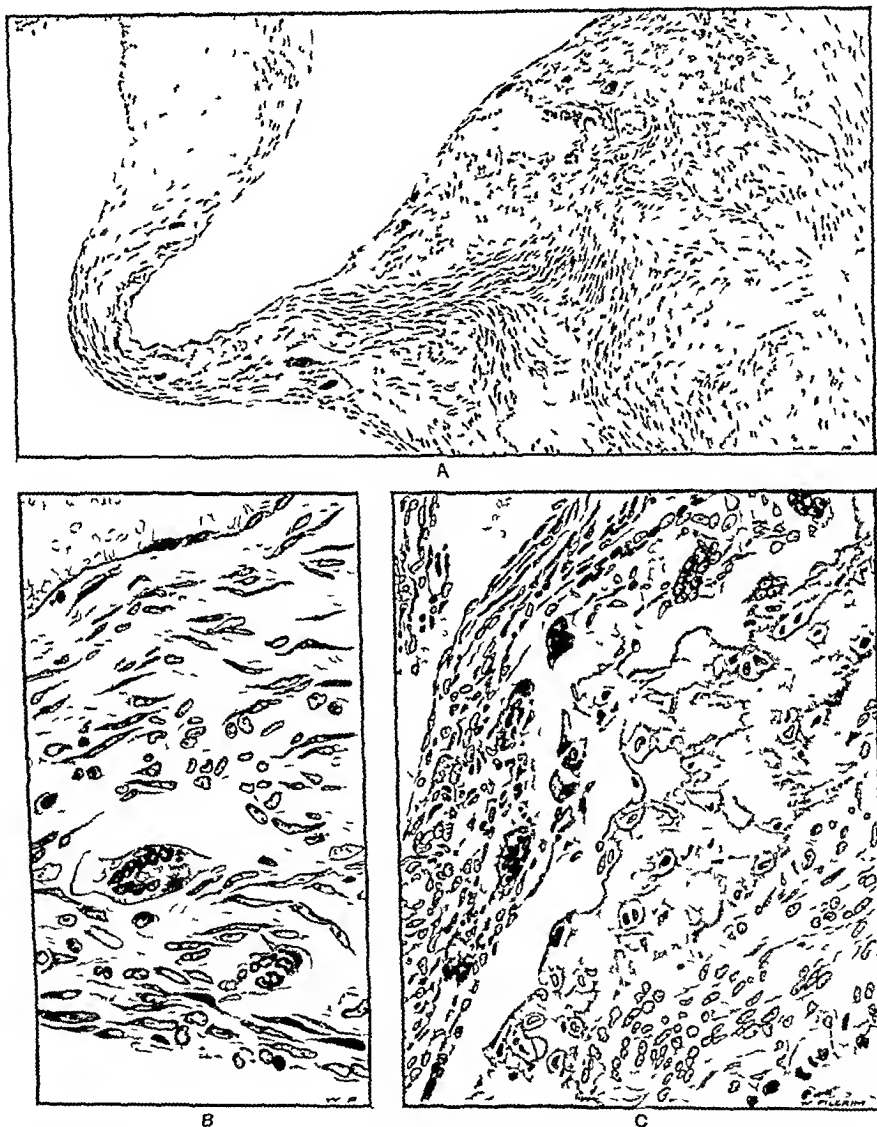


FIG 457—Case 2. Microscopic appearance of tumour shown in Figs 455, 456. A, Low-power drawing of wall of blood-filled cyst in the tumour. B, Higher-power of portion of A, showing cyst wall with osteoclasts. C, Portion of tumour showing erosion of bone by osteoclasts.

SUBSEQUENT PROGRESS—The wound healed by first intention, and convalescence was uninterrupted. The bladder required catheterizing after the operation for eighteen hours and then resumed normal function. There was no trouble with the bowels. The patient was discharged on the forty-ninth day after operation. A special webbing support taking the weight from the right shoulder was designed in consultation with Messrs Ernst and this is worn day and night. Five and a half years after the operation there is no sign of stretching of the abdominal parietes. As a young and active boy he had no difficulty in adapting himself

to new conditions, and was soon able to sit comfortably and to get about with the aid of crutches. He has refused offers of any endeavour to fit a prosthesis. He is now able to take an active part in the work of a busy butcher's shop in his native town, he drives a car, and is at present courting a sweetheart! (Figs 455-458)

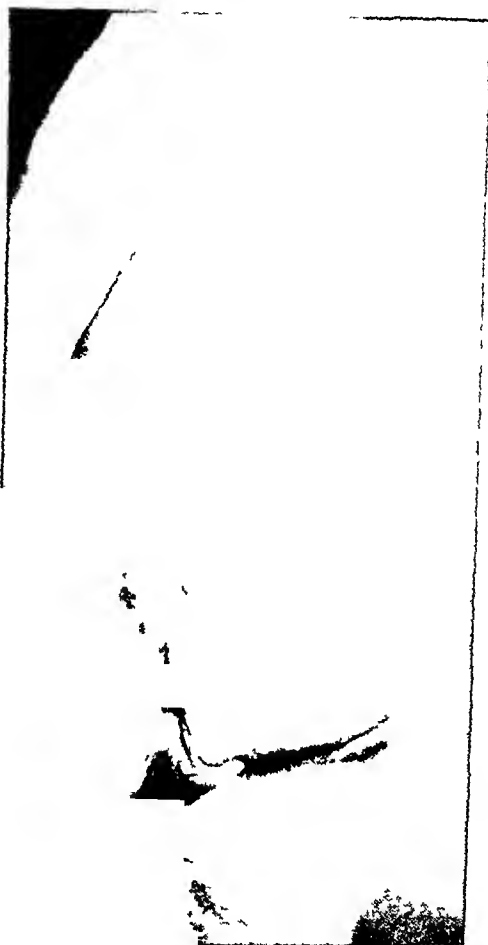


FIG 458—Case 2 Photographs of the patient taken five and a half years after operation

Case 3 —Enormous chondroma of innominate bone, overlapping upper end of femur and extending to costal arch Interinnomino-abdominal amputation Recovery

HISTORY —H. S., male, aged 59, golf-course labourer. The patient had known of the presence of a "lump in the left groin" since infancy. This had gradually increased in size throughout his life, but during the two years preceding his admission the growth of the tumour had been rapid, and it had attained the enormous dimensions shown in the accompanying photographs (Fig 459). The huge size of the mass had interfered with his walking for the past six months, and nine weeks previously he was compelled to give up his work on the golf-course. He could walk less than a hundred yards and had to stop by reason of the pain which he experienced. The pain was of a rheumatic character and radiated down the lateral aspect of the left thigh to the foot, although not always present, this pain was sometimes most severe, and before admission to the Middlesex Hospital its intensity had

necessitated the daily and nightly employment of veramon, nepenthe, etc., in increasing doses for its relief. He was virtually 'house-tied' by reason of his encumbrance and his sufferings.

The patient had been operated upon for a carcinoma of the lower lip in 1931, the growth had been excised and there was *no* evidence of any recurrence.

The enormous tumour of the left innominate bone, for which he claimed admission to hospital, reached to the left costal margin, pushing upwards and outwards the costal outlet, it extended downwards to the great trochanter, anteriorly almost to the umbilicus, and posteriorly it filled up the renal parallelogram. The mass was coarsely nodular, tightly

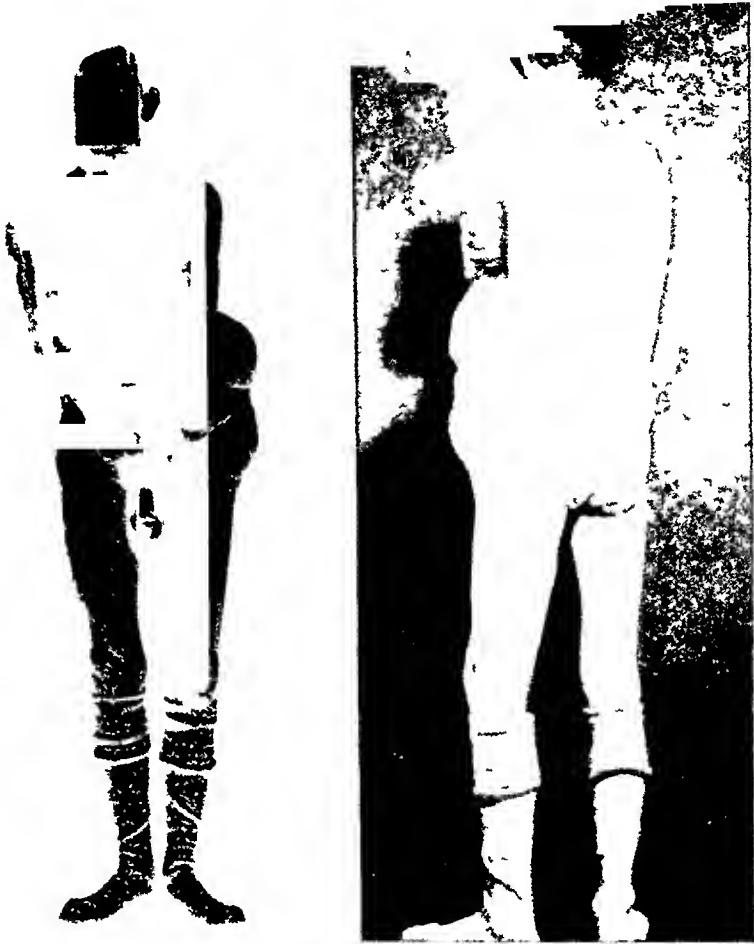


FIG 459—Case 3 Showing the extent of the colossal chondroma of the innominate bone and its upward and downward extension anterior and posterior aspects

stretching the skin, but the latter was not attached to it. The consistence of the tumour varied, in some parts it was cystic, in others very hard. There was well-marked calcification of the blood-vessels of the limbs, as demonstrated by radiography.

The anatomical connections precluded thorough removal of the tumour by any operative measure less severe than a hindquarter amputation, indeed, the manner in which the mass overlapped the great trochanter is well shown in Fig 462.

The rapid growth of the tumour in the recent months which preceded his admission, and the pain experienced, cogently suggested a malignant transformation of an old-standing chondroma, moreover, it was felt that in the event of the tumour not being already malignant in nature, a more local, but imperfect, operation might expose the patient to the supervention



FIG 460—Case 3 Photograph of the patient nearly 1 year after operation



FIG 461—Case 3 Photograph of specimen immediately after removal by hindquarter amputation

of the sarcoma on the relics of a partially ablated chondroma—a calamitous experience which has indeed befallen one of us (G G-T) It was therefore decided that an interinnomino-abdominal amputation held out the best hopes of a permanently successful issue, and, despite the calcified condition of his arteries, arrangements were instituted for its performance

OPERATION (Nov 5, 1933, G G-T)—Messrs Wiles, Banham, and Gowar gave most



FIG. 462—Case 3. Chondroma of innominate bone. The muscles, etc., have been removed and the overlapping of the hip joint and the upper end of the femur by the tumour is well shown. Anterior aspect.

valuable and loyal assistance, and Mr E G Muir made himself responsible for such methods of intravenous therapy as appeared to be indicated during the operation. In point of fact, no less than 1200 c c of blood were transfused during the operation, and in addition 200 c c of saline were infused. Anaesthesia was partly by means of intrathecal injection of 2 c c of 5 per cent stovaine, and partly by means of gas and ether induction with a Clover

inhaler, followed by open ether Mr Idris most skilfully anaesthetized and courageously refused to exhibit any worry or alarm, even when in the middle of the operation the condition of the patient was desperate in the extreme

The position and magnitude of the tumour had occasioned us some anxiety as to the possibility of having sufficient skin-flaps to cover in the large raw area, consequently an

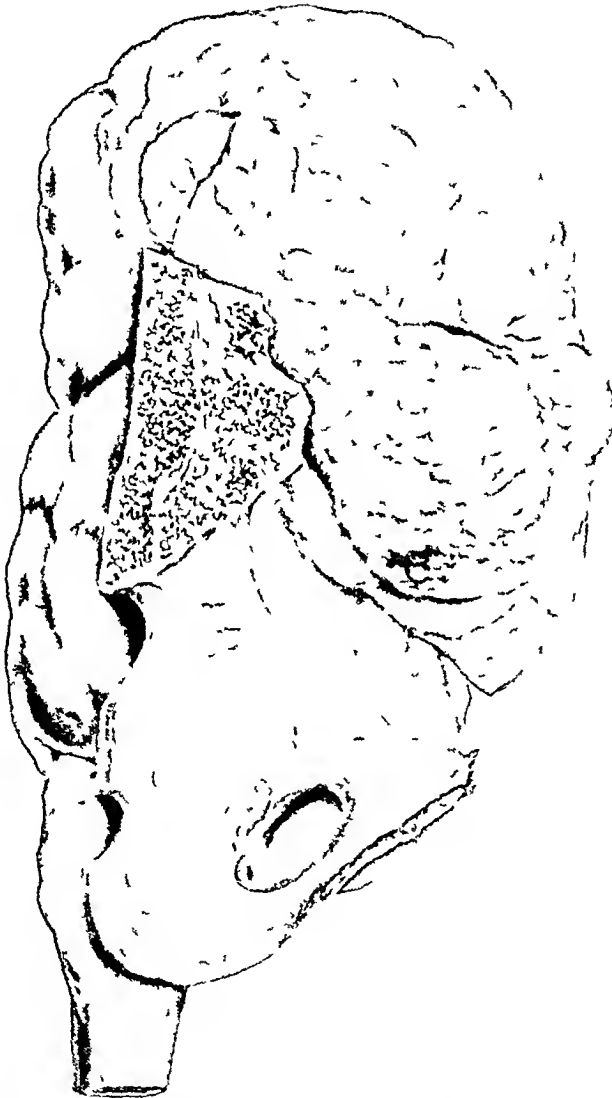


FIG 463—Case 3 Same specimen as shown in Fig 462, posterior aspect The portion of sacrum removed with the innominate bone is shown

internal flap from the adductor region of the thigh was employed according to the method of Savariaud (*see Fig 471, E*) and the superficial femoral vessels were ligated low down so as to endeavour to secure the vitality of this flap Apart from this region of the dissection, where the anatomy was undisturbed, the rest of the amputation was performed blindly, since the colossal tumour had obscured all landmarks, and there was no possibility of demonstrating the branches coming off the internal iliac artery and controlling these before they were cut

The patient had to be moved on to his side and back again into the supine position on more than one occasion, with the result that an alarming fall of blood-pressure ensued which almost proved fatal. Fortunately there was ample blood in readiness for any instant transfusion, and its expeditious and timely employment undoubtedly staved off a disaster that was



FIG 464—*Case 3* Section of specimen shown in Figs 462, 463

imminent. Later in the evening of Nov 5 the patient was given another 500 c c of blood and an additional pint of saline infusion.

SUBSEQUENT PROGRESS—The post-operative convalescence was by no means so uneventful as in *Case 2*, for the internal flap sloughed, and the wound everywhere healed badly. A

post-operative thrombo-phlebitis of the popliteal vein of the other limb also ensued, but despite all these anxieties the patient was able to go to a Convalescent Home on Jan 12, 1934 (Fig 460)

The weight of the dried and macerated tumour, innominate bone, and upper end of femur was 12 lb 4 oz (Figs 461-463) The section shows masses of hyaline cartilage with areas of ossification and cancellous bone (Fig 464)

Case 4—Sarcoma of pelvis Interinnomino-abdominal amputation Death

HISTORY—E L, male, aged 28, had fallen from a lorry eighteen months before his admission into Middlesex Hospital The accident occasioned considerable pain in the vicinity

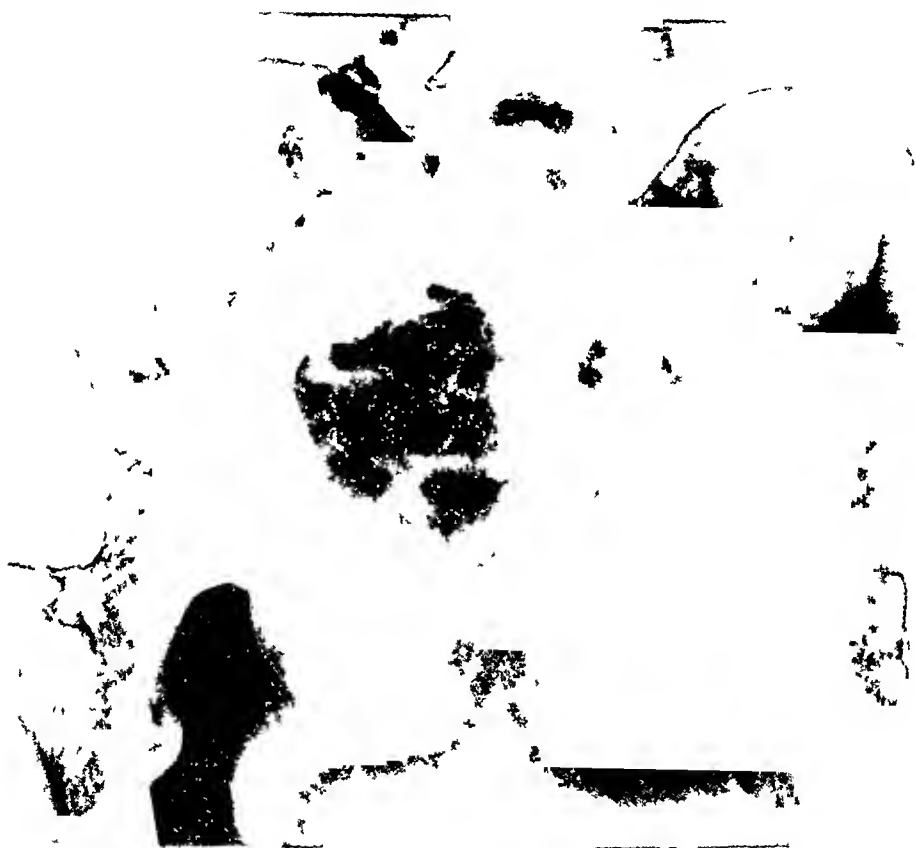


FIG 465—Case 4 Radiogram exhibiting the appearances of sarcoma of the pelvis

of the left hip, which gradually passed off A year ago the pain returned, and although the patient was at first regarded as a case of rheumatism, he was nevertheless X-rayed at a local hospital, where a diagnosis was made of tuberculosis of the left hip-joint He was transferred to a sanatorium and treated by means of rest, splinting, and extension, but the appearance of a swelling in Scarpa's triangle led to the taking of another radiogram, which showed destruction of the ischium and pubes without any new bone formation He was then seen in consultation by Mr Ronald Reid, of Colchester, who at once diagnosed a malignant neoplasm, and he was straightway transferred to my care (G G-T) in Middlesex Hospital

ON EXAMINATION—There was a hard, smooth mass present in Scarpa's triangle It extended down the inner aspect of the thigh for about 4 in and could be felt as an indefinite induration above the mesial end of Poupart's ligament The tumour could also be felt in the ischio-rectal fossa, internal to the ischial tuberosity, there was no alteration in the position

of the anus The left lower limb was maintained in a position of external rotation, the knee was slightly flexed The femoral artery could be felt just lateral to the tumour The case was regarded as a sarcoma, and in the absence of any evidence of metastasis, arrangements were made for amputation

OPERATION (March 3, 1934, G G -T) —Anæsthesia was induced by Mr R E Apperly by means of intrathecal stovaine and a Clover induction, followed by open ether Able assistance was rendered by Messrs P Wiles, Roy Banham, and Alan Kekwick Mr E G Muir kindly made himself responsible for the transfusions, as he had done in the preceding operation (Case 3)

The difficulties of this particular case were due to the fact that the growth had extended

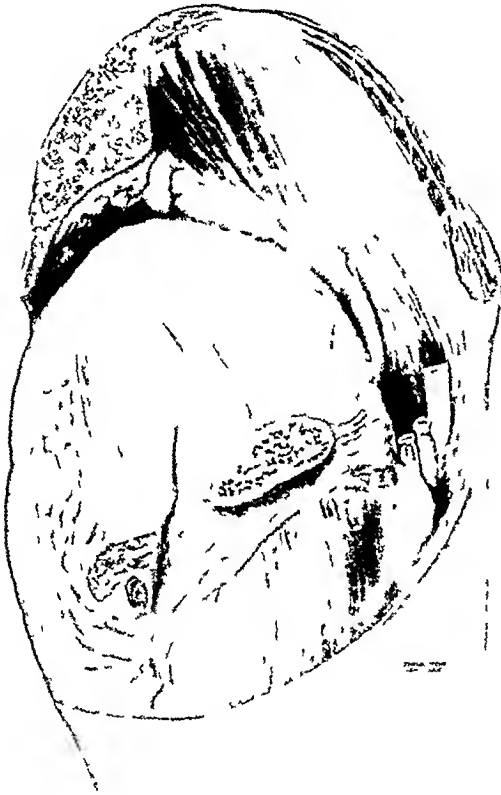


FIG 466—Case 4 Specimen viewed from inner aspect The inward projection of the mass is depicted the latter proved to be somewhat adherent to the rectum and to the prostatic sheath

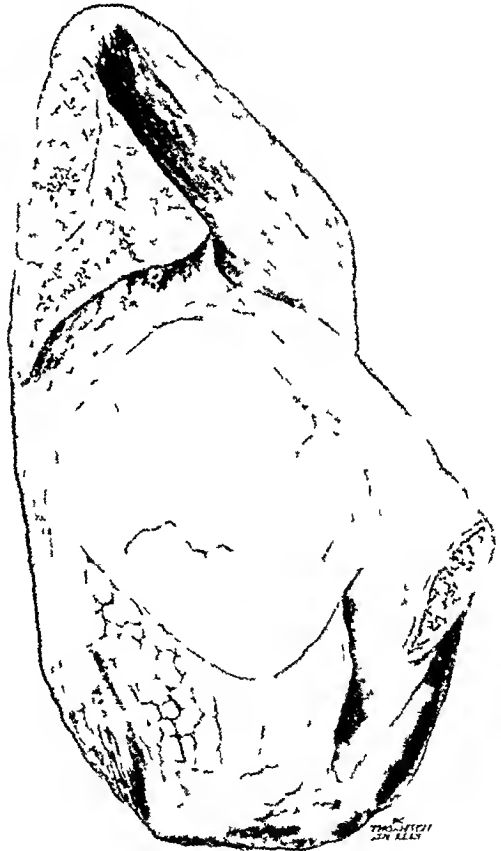


FIG 467—Case 4 Same specimen as shown in Fig 466 on section, exhibiting naked-eye appearance of the neoplasm

beyond the middle line, and the bone section had to traverse the innominate bone of the opposite side The neoplasm was also somewhat adherent to the sheath of the prostate and the rectum, and had to be dissected off these structures Apart from this there seemed nothing very remarkable to note, the operation was completed in sixty-five minutes and the patient left the theatre in good condition A transfusion of 1200 c c of blood from two separate donors was performed during the operation (Figs 465-467)

After his return to bed the patient began to go downhill and a further transfusion was given This was unavailing, and death ensued two-and-a-half hours after the completion of the operation

AUTOPSY—At the autopsy there were no secondary growths anywhere throughout the body. The general musculature of the body was poor and anæmic and the cardiac muscle was especially soft and friable—possibly a consequence of the prolonged sojourn in bed for the treatment of his supposed tuberculosis. Some obsolete tuberculous mesenteric glands were found.

MICROSCOPIC REPORT—The section showed a growth interspersed by strands of fibrous tissue and composed of large hyperchromated cells with scanty cytoplasm. There was very little intercellular substance, *no* alveolar arrangement, and no new bone formation. The appearance was suggestive of a Ewing's endothelial myeloma of bone.

Case 5—Sarcoma of the upper end of the femur Interinnomino-abdominal amputation Recovery

W B, male, aged 18. Admitted for tumour of the upper thigh, which was alleged to have been present for only three weeks.



FIG 468—Case 5. Photograph showing the relation of the tumour to the hip, negating any amputation at that level.

ON EXAMINATION—There was a large fusiform swelling at the junction of the upper and middle thirds of the right thigh, the tumour had almost attained the size of a Rugby football, it was firm but not tender. There were considerable veins coursing under the skin overlying the mass. There were no limitation of the movements of the lower limb (Fig 468).

Radiological Report—At the junction of the upper and middle thirds of the femur there was some rarefaction of the cortex, with periosteal reaction on both sides of the bone. On the posterior surface small spindles could be seen at right angles to the shaft. The appearances were those of a periosteal sarcoma.

The extent of the tumour, its position, and the involvement of the soft parts (Fig 469) excluded any mere disarticulation at the hip-joint as an effective procedure if operation were decided upon, nothing less drastic than an interinnomino-abdominal amputation was likely to cure, if surgery rather than radiation therapy were contemplated. There was no evidence of any secondary growth in any other part of his body.

OPERATION (July 2, 1934, G G-T)—Anaesthesia was by means of intrathecal stovaine (7 cgrm) and also open ether, both were skilfully administered by Mr Raymond Apperly. Messrs P Wiles, Alan Kekwick, and R S Handley rendered able assistance, and Mr Lloyd-Davies kindly made himself responsible for any

intravenous therapy that might be required during the operation.

Operation was simple, inasmuch as the innominate bone was nowhere invaded by new growth, the symphysis was cut through with a knife, the patient was, with the greatest gentleness, turned over toward the sound side, and to a degree that just sufficed to enable the dorsum ilii to be sawn across to the sciatic notch. It was then very easy to secure all the branches of the internal iliac vessels before they were cut, and the nerves were blocked with novocain before being divided. Every step in the operation was taken quickly and quietly, very little shock was produced, but it was deemed prudent at the very end to administer a blood transfusion of 500 c.c. The operation lasted an hour and ten minutes, and the condition of the patient at the end was surprisingly good, reminding all of Case 2 in this series in the excellence of his state at the termination of so severe an amputation. The pulse-rate on the return of the patient to bed was 84. Recovery was uneventful, and the wound healed by first intention.

PATHOLOGICAL REPORT—On section a spindle-shaped tumour arising from the periosteum of the upper half of the femur, chiefly from the inner aspect (transverse diameter, 5 in.) The growth is soft, with areas of hæmorrhage, and is invading the overlying muscles, there is no evidence of bone formation, and little evidence of invasion of the bone cortex. *Spindle-celled sarcoma* (Fig 470)

Section shows infiltration of tissues by solid masses of darkly staining short-spindle cells. There is no evidence of bone formation.

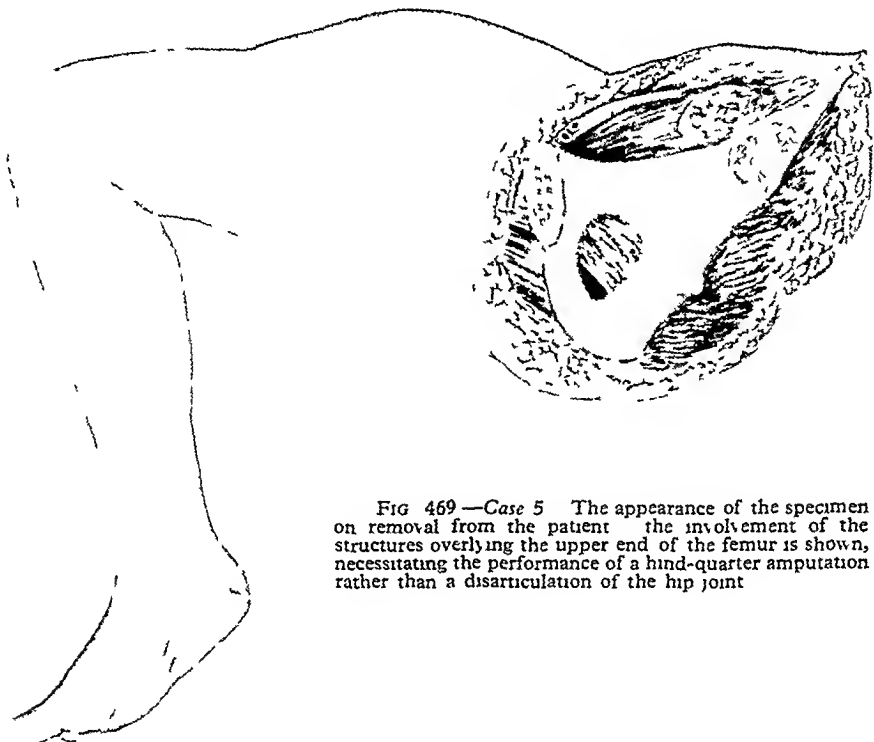


FIG 469.—Case 5. The appearance of the specimen on removal from the patient. The involvement of the structures overlying the upper end of the femur is shown, necessitating the performance of a hind-quarter amputation rather than a disarticulation of the hip joint.

TECHNIQUE OF THE OPERATION

It is significant of the excellence of Hogarth Pringle's description of this amputation that his account³ has been re-read by one of us (G. G.-T.) before the performance of each of the operations on the cases narrated in the preceding pages.

Anæsthesia—In each case spinal anæsthesia was employed in conjunction with general anæsthesia, the latter was induced by means of gas and ether in a Clover inhaler, and this was followed by open ether. *Dosage of stovaine* probably about 7 cgrm. stovaine is an appropriate dosage, but in Case 3 an injection of 10 cgrm. was administered.

Incision—Several incisions have been employed by various surgeons (Fig 471). In four of the cases detailed above the incision was that recommended by Hogarth Pringle; this is a modification of Girard's incision and will be found to satisfy the requirements of most cases, save when the anatomy of the pelvis is grossly deformed by gigantic tumours, such as that depicted in Fig 459 (Case 3). In this case the incision of Savariaud was made use of, and a long

internal flap employed Though every endeavour was made to ensure a good blood-supply to the flap, it sloughed in part Possibly the degenerated state of the arterial system may have accounted for this



FIG 470—Case 5 Specimen of sarcoma of the upper end of the femur on section showing relation to the overlying muscles

Position—The patient is turned slightly on to the sound side, with a sand-bag under the shoulder and another under the lower part of the thigh of the affected side While these dispositions are being made *one of the veins* of the antecubital fossa of

the upper extremity of the sound side is *rapidly exposed*, with a view to facilitate the transfusion of blood, for which previous arrangements have been organized

The incision is carried along the crest of the ilium from behind the posterior superior spine to the anterior superior spine of that bone it is then continued downwards and inwards $1\frac{1}{2}$ in below Poupart's ligament towards the middle of the origin of the adductor brevis Where any doubt is experienced as to the fixity of the neoplasm on the inner aspect of the innominate bone, and as to the possibility of removal, it is well straightway to cut through the flat muscles of the abdominal wall at or above their attachments to the iliac crest, and to satisfy oneself at once as to the pathological integrity of the peritoneum, etc., before proceeding to deal with the external iliac vessels Where no such doubts are felt, it may be of some comfort to the operator to deal with the external iliac vessels

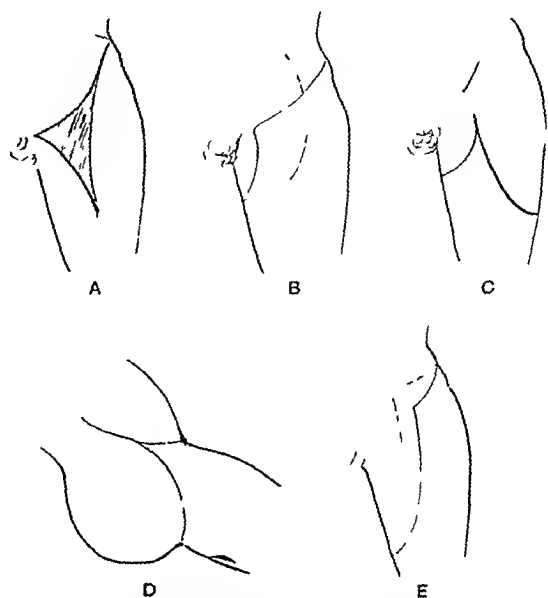


FIG 471 —Diagram showing some of the various incisions that have been employed in the operation of interinnomino abdominal amputation A, Jaboulay, B, Guard, C, Bardenheuer, D, Salistcheff, E, Savariaud

as soon as this first incision is made Except in Case 3, where the femoral vessels were ligated, the external iliacs have been secured at the level of Poupart's ligament No direct search was made for deep epigastric or deep circumflex iliac vessels, and they do not appear to have been damaged

Poupart's ligament is divided at each end (Fig 472), and the inner pillar of the external abdominal ring, thus allowing the spermatic cord to be drawn inward out of harm's way The insertion of the rectus abdominis to the pubic crest is next divided, and, after the anterior and posterior surfaces of the pubic bone have been cleared by blunt dissection the symphysis pubis is divided by knife, or if necessary by saw (Fig 473)

The patient is then very gently turned further over on to the sound side, and an incision carried from the mid-point of the iliac crest down to the gluteal fold, which is followed to meet the lower and mesial extremity of the first incision in the cruro-scrotal sulcus The dorsum ilii is exposed and sawn through into the sciatic

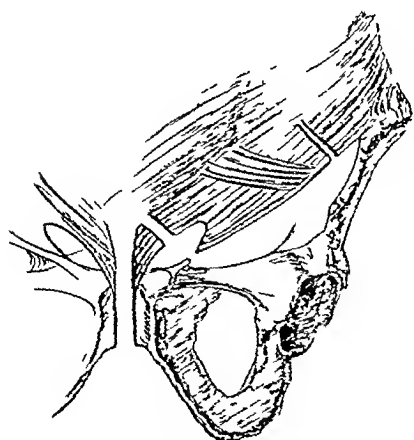


FIG 472 —Diagrammatic sketch indicating one stage in the operation Poupart's ligament has been divided at each end, and the inner pillar of the external abdominal ring has been cut through enabling the operator to retract the spermatic cord inwards and to get access to the insertion of the rectus abdominis The rectus and the symphysis are now divided

followed to meet the lower and mesial extremity of the first incision in the cruro-scrotal sulcus The dorsum ilii is exposed and sawn through into the sciatic

notch this manœuvre is more simple and expeditious than attempting to disarticulate at the sacro-iliac synchondrosis. If it be necessary, the small piece of the auricular surface of the ilium can be rapidly detached afterwards.

The anterior and posterior bone sections being now complete, the innominate bone and lower extremity can be drawn away from the pelvic peritoneum and its contents (*Fig. 474*).

The lumbo-sacral cord, the first and second sacral nerves, and the obturator can all be identified and injected with novocain before division. The obturator artery, the gluteal artery, the sciatic artery, and probably the internal pudic artery, can be secured before division, and in this manner the hæmorrhage can be greatly minimized.

The psoas is cut through above the pelvic brim, and the division of the pyramiformis and the levator ani, and the detachment of the ischiocavernosus and crus penis from the ischiopubic ramus liberates the innominate bone from the last fibromuscular links which fetter it to the underlying structures, the limb and the innominate bone are free.

All bleeding is now controlled, and the peritoneum reinforced by the suture of the remains of the gluteus maximus, levator ani, etc., to the flank muscles and rectus abdominis. The skin is then sutured with interrupted stitches.

Retention of urine has followed the operation in the three of our cases that survived, and catheterization has been required.

The most important points in the conduct of the operation would appear to be (1) The most gentle care should be taken in turning the patient: the danger from rough or excessive movement of a patient under spinal anæsthesia cannot be exaggerated. (2) The most thorough organization of blood transfusion arrangements. (3) Division of the posterior portion of the dorsum ili with the saw is more expeditious and simple than disarticulation of the bone at the sacro-iliac synchondrosis, it is easy subsequently to ablate the auricular portion of the ilium. (4) Hogarth Pringle's incision is that most universally satisfactory. (5) It is almost unnecessary to stress the importance of minimizing hæmorrhage and shock by preliminary control of vessels before division, and of nerve-blocking before section of the trunks.

STATISTICS

One of us (P. W.) has expended much time in bringing the literature of the subject up to date, and, including the 5 personal cases placed on record in this communication, has verified the references in 55 cases in which the innominate

Table I.—OPERATIVE MORTALITY IN INTERINNO-MINO-ABDOMINAL AMPUTATION

NATURE OF DISEASE	NUMBER OF CASES	RECOVERY	DEATH	PERCENTAGE MORTALITY
Sarcoma	37	17	20	54.1
Tuberculosis	15	5	10	66.6
Non-tuberculous disease of innominate bone and hip-joint	1	1	—	—
Osteoclastoma	1	1	—	—
Unknown origin	1	—	1	—
Total	55	24	31	56.4

INTERINNOMINO-ABDOMINAL AMPUTATION 691

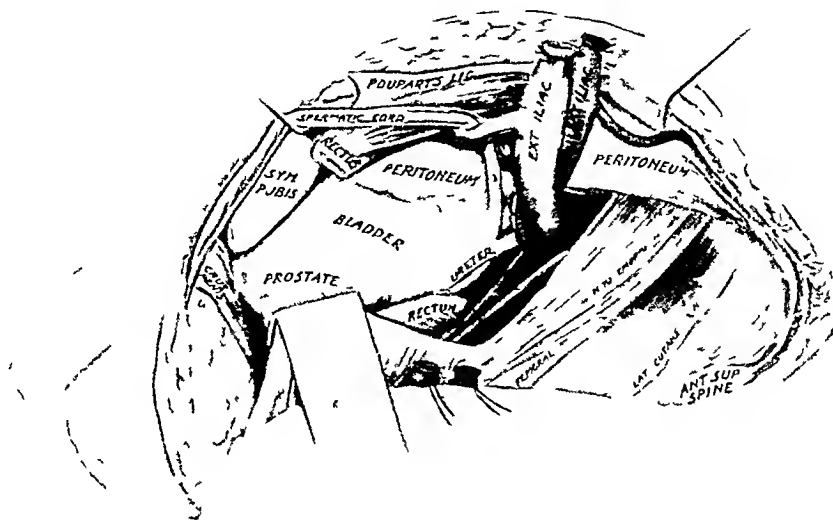


FIG 473—Anatomical structures displayed during interinnomino-abdominal amputation. The external iliac vessels have been ligatured distal to the origin of the epigastrics, the symphysis has been divided, and the innominate bone is being retracted outwards.

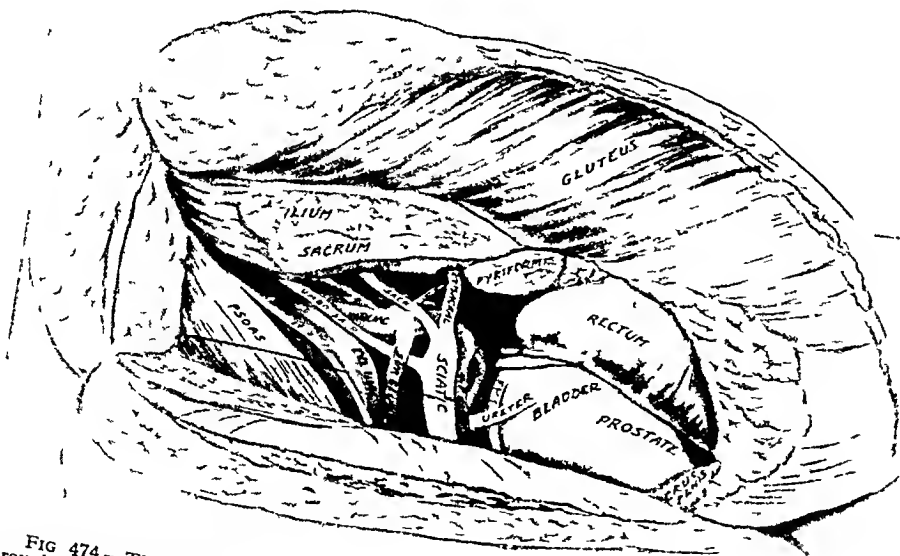


FIG 474—The patient has been turned over on to the sound side and the dorsum illi has been sawn through the symphysis pubis having been divided, the innominate bone is being drawn away from the pelvic peritoneum and the pelvic viscera. The arteries and nerves which require surgical attention or division are shown. The line of section of the psoas is indicated.

Table II—DETAILS OF INTERINNUMINO-ABDOMINAL AMPUTATIONS RECORDED IN THE LITERATURE (55 CASES)

SURGEON AND DATE	SEX AND AGE	DISEASE	RESULT	REMARKS
Abrashanaw, A (1930)	F 35	Sarcoma	Lived at least 3 months	Reported by S I Riswach (<i>Deut Zeits f Chir</i> , 1932-3, cxxxviii, 121) Momburg's tourniquet Cut ilium and pubis and joined with graft (<i>Arch f klin Chir</i> , 1910, xci, 538) <i>Zentralb f Chir</i> , 1897, xxiv, suppl, 132, also <i>Verh Deut Gesel f Chir</i> , 1897, xvi, 130 Reported by O Wolff, <i>Zentralb f Chir</i> , 1897, xxiv, 185 Three previous local operations (<i>Surg Gynecol and Obst</i> , 1918, xvi, 554) Momburg's tourniquet (<i>Deut med Woch</i> , 1909, xxiv, 1, 45) Savariaud says that Berg, of Stockholm, told Girard he was assistant to Billroth when he performed this operation <i>Riforma Med</i> , 1894, ii, 819 and 831 Reported by Dreist (<i>Deut Zeits f Chir</i> , 1904, lxxi, 4) Temporary ligature aorta Operation abandoned because of hemorrhage (<i>Rev de Gynecol et de Chir abdom</i> , 1899, iii, 713) First disarticulated hip, then removed a lot of pelvis (<i>Ann of Surg</i> , 1901, xxxiii, 318) Disarticulation of hip 2 years before (<i>Four de Chir (Belge)</i> , 1901, i, 569) Disarticulation of hip 2 years before (<i>Ibid</i>) Momburg's tourniquet Disarticulated hip-joint (<i>Bull et Mem Soc Chir de Paris</i> , 1913, xxxv, 1570) Disarticulation of hip the year before (<i>Congres fran de Chir</i> , 1895, i, 823) Previously had resection of knee (<i>Ibid</i> , 1898, vii, 585) <i>Ibid</i>
Axhausen, O (1910)	F 45	Sarcoma	Died in 9 hours	
Bardenheuer (1897)	F 46	Tuberculosis	Alive 10 months later	
Babcock, W W (1916)	M 52	Sarcoma	Died of shock soon after operation	
Bier (1909)	>	Sarcoma	Lived 2 months	Some months after operation had a child with easy labour (<i>Surg Gynecol and Obst</i> , 1926, xliii, 668) Patient in extremis at time of operation (<i>Lyon med</i> , 1894, lxxv, 507) Reported by G Gayet (<i>Proence med</i> , 1895, xxiv, 397) Poor condition metrastrases found post mortem (<i>Ljstopsis Russ Chir</i> , 1900, iv, and in <i>Fahrh.</i>)
Billroth (1891)	>	>	Died in few hours	
Caccopoli, G (1894)	M 17	Sarcoma	Died of shock in 3 hours	
Christel (1903)	F 2	Tuberculosis	Lived	
Faure, J L (1899)	F 16	Sarcoma	Died next day	Some months after operation had a child with easy labour (<i>Surg Gynecol and Obst</i> , 1926, xliii, 668) Patient in extremis at time of operation (<i>Lyon med</i> , 1894, lxxv, 507) Reported by G Gayet (<i>Proence med</i> , 1895, xxiv, 397) Poor condition metrastrases found post mortem (<i>Ljstopsis Russ Chir</i> , 1900, iv, and in <i>Fahrh.</i>)
Freeman, L (1899)	F 38	Sarcoma	Lived 20 months	
Gallet (1900)	M 25	Tuberculosis	Died in 6 hours	
Gallet (1901)	F 39	'Telangiectatic' sarcoma	Died in 1 hour	
Gaudier, H (1913)	M 15	Non-tuberculous osteomyelitis	Lived	Some months after operation had a child with easy labour (<i>Surg Gynecol and Obst</i> , 1926, xliii, 668) Patient in extremis at time of operation (<i>Lyon med</i> , 1894, lxxv, 507) Reported by G Gayet (<i>Proence med</i> , 1895, xxiv, 397) Poor condition metrastrases found post mortem (<i>Ljstopsis Russ Chir</i> , 1900, iv, and in <i>Fahrh.</i>)
Girard (1895)	F 17	Sarcoma	Lived, 7 months later	
Girard (1895)	M 15	Tuberculosis	Died in 5 minutes	
Girard (1897)	M 52	Sarcoma	Lived 6 months and died of secondaries	
Gordon-Taylor, G (1922)	M 25	Sarcoma	Died of shock in 6 hours	Some months after operation had a child with easy labour (<i>Surg Gynecol and Obst</i> , 1926, xliii, 668) Patient in extremis at time of operation (<i>Lyon med</i> , 1894, lxxv, 507) Reported by G Gayet (<i>Proence med</i> , 1895, xxiv, 397) Poor condition metrastrases found post mortem (<i>Ljstopsis Russ Chir</i> , 1900, iv, and in <i>Fahrh.</i>)
Gordon-Taylor, G (1929)	M 17	Osteoclastoma	Lived 5½ years later	
Gordon-Taylor, G (1933)	M 59	Chondrosarcoma	Lived	
Gordon-Taylor, G (1934)	M 28	Sarcoma	Died in 2½ hours	
Judin, S S (1924)	M 18	Sarcoma	Lived	Some months after operation had a child with easy labour (<i>Surg Gynecol and Obst</i> , 1926, xliii, 668) Patient in extremis at time of operation (<i>Lyon med</i> , 1894, lxxv, 507) Reported by G Gayet (<i>Proence med</i> , 1895, xxiv, 397) Poor condition metrastrases found post mortem (<i>Ljstopsis Russ Chir</i> , 1900, iv, and in <i>Fahrh.</i>)
Jaboulay (1894)	F 36	Chondrosarcoma	Lived at least 2½ years	
Jaboulay (1895)	>	Sarcoma	Died	
Jaboulay (1895)	M 66	Sarcoma	Died 36 hours from infection	
	M 25	Sarcoma	Died of shock in 2 days	

Lastaria, F (1906) Loeffler, F (1918) Maffei, A (1915)	M 22 M 11 F 5	Chondrosarcoma Sarcoma Tuberculous hip	cacnevix Died during operation Lived at least 15 months Lived Well 17 years after	in <i>Zentralbl f Chir</i> , 1910, xxxvii, 1132 <i>Reforma Med</i> , 1907, xxii, 457 <i>Zeits f orthop Chir</i> , 1919, xxxix, 305 Numerous fistulae, high temperature burg's tourniquet X-rays and photographs taken in 1932 (<i>Proc Soc Internat de Chir orthop</i> , 1933, 270) Recovered from immediate effects of opera- tion Temperature did not fall, disease continued its course "Le Traitement de la Coxite tuberculeuse", by A Maffei (Bruxelles) 2 ^{me} Congrès <i>internat de Chir orthop</i> , Londres, 1933, p 270 Operation abandoned before finished (<i>Bull Soc Anat de Paris</i> , 1902, iv, s 6, 795) Amputation of hip 1 year before Very emaciated Open sinuses (<i>Bull Soc Chir Paris</i> , 1908, xxxiv, 1060) Leg amputated at hip 2 months earlier In this and 1905 case the tuber ischi was left (<i>Bull Soc Anat de Paris</i> , 1909, xi, s 6, 288 <i>Congrès internat de Med</i> , Paris, 1900 (Sec Chir gen), 511
Maffei, A (1915)	M 8	Tuberculous hip, cachectic, fistulae	Lived one month	
Maffei, A (1915)	?	(Child) tubercu- lous hip	Died in 2 hours	
Morestin (1902)	F ?	Sarcoma	Died in 9 hours	
Morestin (1905)	M ?	Tuberculosis	Lived at least 3 years	
Morestin (1909)	M 35	Tuberculosis	Died in 5 hours	
Nanu (1900)	M 50	Myxosarcoma	Died in 20 days of gangrene flaps—tied common iliac vessels	
Orlow, L (1907)	M 47	Sarcoma	Died 35th day of sepsis of wound	<i>Westnik Chir</i> , 1907, ii, 225, abstr in <i>Jahrb v d Forts d Chir</i> , 1907, vii, 1104
Pagenstecher, E (1909)	F 50	Sarcoma	Lived at least 3 months	Momburg's tourniquet (<i>Arch f klin Chir</i> , 1909, xc, 160)
Roux (1909)	M 40	Sarcoma	Lived at least 3 months	Reported by G Plantard (<i>Arch Prov de Chir</i> , 1909, xviii, 657)
Pringle, H (1900)	F 10	Tuberculosis	Lived 14 years	Amputation of hip and removal of much of pelvis (<i>Lancet</i> , 1909, i, 530)
Pringle, H (1906)	M Adlt	Tuberculosis	Died in 14 hours	Multiple sinuses (<i>Ibid</i>)
Pringle, H (1908)	M 18	Sarcoma	Lived 5 months, died of secondaries	Amputation of leg 2 years before (<i>Ibid</i>)
Pringle, H (1915)	M 34	Sarcoma	Lived	<i>Brit Jour Surg</i> , 1916, iv, 283
Pringle, H (1915)	F ?	Sarcoma	Lived	<i>Ibid</i>
Ransohoff, J (1909)	M 45	Sarcoma	Died in 4 weeks from 'entero-colitis'	Poor condution, multiple sinuses (<i>Ann of Surg</i> , 1909, l, 925)
Ribera, Y S (1902)	?	Tuberculosis	Died in 8 days from gan- grene of flaps Tied common iliac artery	Reported by Lous y Simon, <i>Siglo med</i> , 1903, l, 779
Ribera, Y S (1902)	?	Tuberculosis	Died soon after operation	<i>Ibid</i>
Ribera, Y S (1902)	F 10	Tuberculosis	Died 3 hours after operation	<i>Ibid</i>
de Ruyter (1902)	M 45	Sarcoma	Died in 1 hour	H Mayer, <i>Inaug Dissert</i> , Leipzig, 1902
Schistcheff (1898)	M 38	Sarcoma	Lived Tied common iliac artery, healing by second intention	<i>Arch f klin Chir</i> , 1900, ix, 57
Savarnaud (1901)	F 7	Sarcoma	Died same day of 'syncope'	<i>Rev de Chir</i> , 1902, xxvi, 345
Schadclmose, V (1923)	?	Chondrosarcoma	Lived at least 10 months	<i>Acta Scand Chir</i> , 1924, lvi, 523 Momburg
Speed, Kellog (1932)	M 59	Sarcoma	Died at end of operation	Secondaries lungs (<i>Ann of Surg</i> , 1932, xcv, 167)
Speed, Kellog (1932)	M 45	Sarcoma	Died in 1 hour	<i>Ibid</i>
Verneuil (1905)	?	Tuberculosis	Died in 2 hours	<i>Jour de Chir et Ann Soc belge de Chir</i> , 1905, v, 406 Cited by Pringle, <i>Brit Jour Surg</i> , 1916, iv, 283

Table III—CHRONOLOGICAL TABLE OF OPERATIONS AND SURFONS

1891	Billroth	Died	1907	Orlov	Died
1894	Cacciopoli	Died	1908	Pringle	Lived
	Jaboulay	Died		Krynski	Lived
1895	Girard	Lived	1909	Bier	Lived
	Girard	Died		Morestin	Died
	Jaboulay	Died		Pagenstecher	Lived
1897	Bardenheuer	Lived		Ransohoff	Died
	Girard	Lived		Roux	Lived
1898	Salistcheff	Lived	1910	Auxhausen	Died
1899	Faure	Died	1913	Gaudier	Lived
	Freeman	Lived	1915	Pringle	Lived
1900	Gallet	Died		Pringle	Lived
	Nanu	Died		Maffei	Lived
	Pringle	Lived		Maffei	Died
1901	Gallet	Died		Maffei	Died
	Kadjan	Died	1916	Babcock	Died
	Savariaud	Died	1918	Loeffler	Lived
1902	Morestin	Died	1922	Gordon-Taylor	Died
	Ribera	Died	1923	Schadelmoser	Lived
	Ribera	Died	1924	Judin	Lived
	Ribera	Died	1929	Gordon-Taylor	Lived
	de Ruyter	Died		Riswach	Lived
1903	Christel	Lived	1930	Abrashanaw	Lived
	Keen	Died	1932	Speed	Died
1905	Morestin	Lived		Speed	Died
	Verneuil	Died	1933	Gordon-Taylor	Lived
1906	Lastaria	Died	1934	Gordon-Taylor	Died
	Pringle	Died		Gordon-Taylor	Lived

Table IV—CASES FROM THE "RUSSIAN LITERATURE"

SURGEON AND DATE	SEX AND AGE	DISEASE	RESULT	REMARKS
Orlov (1901)	M 47	Sarcoma	D	<i>Messenger of Surgery</i> (Russian), 1901, No 2
Michailow (1902)	M 17	Tuberculosis	D	<i>Surgery</i> (Russia), 1902, v, 11
Sinakevitch (1908)	M 47	Sarcoma	D	<i>Surgery</i> (Russia), xlvii, p 60
Veinshall (1921)	M 25	Sarcoma	D	<i>New Surg Arch</i> (Russia), iii, 147
Bergman (1918)	? ?	5 non-T B osteomyelitis	2 D, 3 L	Quoted by Judin from Veinshall
Bryosovskii (1921)	F 16	Sarcoma	L	<i>New Surg Arch</i> , iv, 179
Czerny } Goldberg }				Cited by Bryosovskii, no details given or results
Israel	F ?	Sarcoma	D	Cited by Bryosovskii
Smirnov (1919)	M 33	Tuberculosis	D	Cited by Bryosovskii
Toprover-Krimov	M 47	Sarcoma	L 2 months	<i>Ekaterinoslav Med Jour</i> , 1924, 1, 44
Rabinovitch	M 58	Sarcoma	L 5 weeks	<i>New Surg</i> , 1925, iii, 374
Rabinovitch	M 49	Sarcoma	D	<i>New Surg</i> , 1925, iii, 374
Krassintzev	M 43	Sarcoma	L	<i>Moscow Surg Soc</i> , 1925, xii

D = Died L = Lived

Table V—PERSONAL COMMUNICATIONS TO JUDIN

Kokin	F 30	Sarcoma	Died
Rosanol	F 27	Tuberculosis	Died
Rosanol	M 35	Sarcoma	Lived 3 months
Rosanol	M 30	Sarcoma	Died
Krassintzev	M ?	Sarcoma	Died
Krassintzev	M ?	Sarcoma	Died
Krassintzev	M ?	Sarcoma	Died
Krassintzev	M 45	Sarcoma	Died

bone and lower limb have been removed, either as a single-stage or as a plurigrade amputation. In some of the cases included in this series the removal of the innominate bone has been incomplete.

Of these 55 cases, 43 complete amputations have been performed as a primary operation. These cases are analysed in *Table I*.

It will be seen that the mortality of the procedure in the cases that we have been able definitely to verify, has been 56.4 per cent. *Tables II* and *III* give details of cases found in the literature.

A close scrutiny of the 74 cases reported by Judin⁴ in 1926 reveals the fact that no fewer than 9 of these cases were duplicates, that another patient is reported to have died whereas the case ended successfully, and there are other inaccuracies which need not be elaborated here. A certain lack of confidence is naturally created, and it becomes difficult to assess the degree of credence that should be given to those reports from the "Russian literature", to which we ourselves have been unable to gain access (*Tables IV, V*).

If we add these "Russian figures" to those that we have confirmed and verified, there are some additional 24 cases with 16 deaths, 16 of these operations were for sarcoma, of whom 11 succumbed to the operation.

The operation has therefore been done about 79 times, with 46 deaths, i.e., a mortality-rate of 59.5 per cent.

In judging these figures it must be remembered that unsuccessful cases are much less likely to see the light of day than are successful ones, this statement is more particularly true at the present time, when the operation is an established procedure, than formerly, when even an attempted operation of this character was indeed an achievement of note. It is almost certain, therefore, that the mortality-rate given here underestimates the real gravity of the operation.

SUMMARY

1 A short note is given of five interinnomino-abdominal or hindquarter amputations, of which three were successful. Of the two fatal cases, one undoubtedly died from a failure in the transfusion arrangements, such as would not occur to-day, in the other fatal case, death appeared to have been due to cardiac failure. In this patient it was perhaps not sufficiently appreciated how baneful an effect upon the heart muscle and on the tissues of the body had been the very long confinement to bed on a mistaken diagnosis.

2 The operation will always remain one of the most colossal in surgery, but organization of team-work and the perfecting of transfusion arrangements will bring the mortality of the amputation down to a still lower percentage.

3 An attempt has been made to bring the literature of the operation up to date.

4 A description of the operation has been included.

For the beautiful pictures which adorn this paper we have to thank our friend Mr Thornton Shiells.

REFERENCES

- ¹ BILLROTH, Quoted by Savariaud, *Rev. de Chir.*, 1902, *xxvi*, 345.
- ² GIRARD, *Congres. franç. de Chir.*, 1895, *ix*, 823.
- ³ PRINGLE, H., *Brit. Jour. Surg.*, 1916, *iv*, 283.
- ⁴ JUDIN, *Surg. Gynecol. and Obst.*, 1926, *xlvi*, 668.

THE PATHOLOGY OF CONGENITAL GENU RECURVATUM*

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THE striking deformity which has been called in the past 'congenital dislocation of the knee' or 'luxation congenitale du tibia en avant', but which is now more accurately described as 'congenital genu recurvatum', has been known to surgeons and orthopædists since 1822, when, according to Potel, Chatelain, a Swiss observer, placed a case on record. I have been unable to verify the reference personally (*Bibliothèque médicale*, 1822, lxxv, 103). Since that time many cases have been described, and in 1897 Potel⁶⁴ gave a masterly description of the malformation in his thesis on deformities of the knee. At that date he was able to give abstracts of no fewer than 78 cases, which constituted the literature of the subject since the first case recorded by Chatelain. Since the publication of Potel's thesis nothing appears to have been added to our knowledge of the pathological processes involved in the production of the deformity, though I have been able to collect *personally* verified references to 80 fresh cases, giving a total of 158 cases.

Briefly, the malformation has in all recorded cases consisted of a fixed hyper-extension of the leg at the knee-joint. The anterior aspect of the joint shows transverse creases in the skin, and the patella is small or may be absent. Posteriorly the hamstring muscles appear to be stretched and the femoral condyles may be felt projecting in the popliteal fossa. The deformity may be increased to some extent by manipulation, but on attempting to flex the joint a distinct elastic resistance is appreciated. In some of the cases on record manipulation succeeded in overcoming the resistance without much difficulty, but it should be noted that this occurred before the introduction of radiography, and it will appear from the case I am about to describe that the reposition which some of the older authors claimed may have been only apparent. In the majority of cases a slight degree of flexion can be produced by manipulation, but on releasing the limb the leg at once springs back into the abnormal position. In the few cases where dissection has been carried out it has been noted that the articular surfaces are invariably normal in every respect. Tarnier,⁸⁶ indeed, observed that the stretched hamstring muscles might pass anterior to the femoral condyles and act as extensors at the knee-joint. It is certain, however, that this is the result of the deformity and not its cause.

The deformity may be unilateral or bilateral. It may be accompanied by isolated contractures elsewhere or by more or less generalized contractures of the limbs. It shows an especial tendency to be associated with club-foot and with congenital dislocation at the hip of the deformed limb.

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ETIOLOGY

Three main theories have been advanced to explain the malformation

1 **The Theory of Uterine Compression**—It is not perhaps unnatural that it should have been supposed that fixation of the knee-joint with the leg in hyperextension during intra-uterine life, and with the foot of the foetus hooked over the shoulder, was responsible for the fixed hyperextension persisting into post-natal life. Uterine compression has for many years been blamed for almost every known congenital deformity, though there is clear evidence in the writings of the last generation that such a theory has failed to satisfy those who had seriously considered the question. I have had the opportunity elsewhere of stating my reasons for believing that uterine pressure is never the cause of real and persistent deformities.⁵⁴

2 **The Theory of Epiphysial Separation**.—Phocas⁶² believed that the deformity was not present actually at the joint, but that the hyperextension resulted from a forward displacement of the distal femoral epiphysis resulting from intra-uterine injury. His theory was accepted for a short time and good results were recorded from "manual osteoclasis of the femur". Dissections and the advent of radiography have proved his theory to be wrong.

3 **The Theory of Muscular Contracture**.—Potel⁶⁴ arrived at almost complete understanding of the problem when he pointed out that the hyperextension was entirely due to contracture of the quadriceps extensor muscle group gradually dragging the knee into a deformed position during intra-uterine growth. He pointed out that in all severe cases tenotomy of the quadriceps tendon was necessary before reduction could be obtained, and suggested that the 'manual osteoclasis' of the older authors was merely a subcutaneous rupture of the quadriceps or patellar tendons. Owen,⁶⁰ in 1891, had the distinction of being the first surgeon to carry out an open division of the quadriceps tendon in order to bring about reduction, and Potel in discussing this case makes the following remark: "If the old adage be true '*Naturam morborum ostendunt curationes*', this single observation suffices to show that the cause of the hyperextension lies in the triceps (quadriceps) muscle."

Potel believed when he first studied the malformation that the muscular contracture was spasmodic in origin, and later⁶⁶ that it was due to simple arrest of development of the muscle. I have recently been enabled to observe a case of this relatively rare deformity which, I believe, sheds fresh light upon the cause of the quadriceps contracture.

CASE REPORT

An otherwise healthy female child, aged 5 weeks, was born with a deformity of the right leg. The infant was the firstborn of healthy and unrelated parents, there was no history of deformity occurring in any blood relative, and the pregnancy and labour had been uneventful.

ON EXAMINATION—A definite degree of hyperextension at the right knee was evident (Figs 475, 476), with two transverse furrows occupying the angle in front of the joint. The femoral condyles were easily palpable as projections in the popliteal fossa. The thigh appeared slightly wasted, but it was difficult to estimate the degree with accuracy owing to a plentiful deposit of subcutaneous fat.

On attempting to flex the knee an elastic resistance was felt and was clearly due to shortening of the quadriceps extensor apparatus. On releasing the leg the knee-joint at once returned to the hyperextended position. Radiography showed the hip-joints to be normal.

The infant was admitted to the Church of Scotland Deaconess Hospital and manipulative reduction was twice attempted under anæsthesia. On both occasions it was found that by steady pressure flexion to about 90° could be obtained, but subsequent radiography showed



FIG 475—Congenital genu recurvatum in a female of 18 months



FIG 476—Radiogram at 18 months showing hyperextension deformity of the knee joint



FIG 477—Radiogram at the age of 6 weeks to illustrate the pseudo reduction produced by manipulation. The popliteal surface of the tibia lies in contact with the articular aspect of the femur.

that the reduction was a false one, the popliteal aspect of the tibia lying in contact with the articular aspect of the femoral condyles (Fig 477).

At the age of 18 months the hyperextension deformity was still present, though a striking change in the contour of the tibia had taken place, this bone being curved in its upper third with a decided anterior convexity, the apex of the curve coinciding roughly with

the attachment of the patellar tendon to the tibial tuberosity. This secondarily acquired deformity is of great interest, as it demonstrates the striking effect which abnormal muscular pull may play in the production of bony deformities (*Figs 476, 478*)

OPERATION—Operative reduction of the subluxation was now undertaken. A vertical incision was made over the antero-lateral aspect of the knee-joint and was carried well up the thigh. The incision was deepened to expose the quadriceps muscle and the knee-joint. The quadriceps muscle was found to be atrophied, and appeared to the naked eye to be composed almost entirely of fat and fibrous tissue. Division of the quadriceps tendon and of its lateral expansions at once liberated the proximal extremity of the tibia, which slipped, under the influence of gravity alone, into its normal relationship to the condyles of the femur (*Fig 478*). Several portions of tissue were removed from the quadriceps for microscopical examination and the wound was closed.

PATHOLOGY—Sections of the quadriceps muscle showed small areas of normal muscular tissue embedded in large tracts of fat and fibrous tissue. In some parts very fine atrophied fibres are visible and many normal muscle spindles can be seen standing out from the surrounding fat and fibrous tissue.

(*Figs 479-481*) No definite immature fibres at the myoblastic stage of development were recognized, though in places the atrophied fibres had a rather similar appearance.

SUBSEQUENT PROGRESS—On re-examination when the child was 3 years and 3 months old, it was seen that the deformity remained reduced and a surprising degree of stability existed in the joint, the child, who was learning to walk, being able to bear a considerable amount of weight upon the affected limb.



FIG 478—Radiogram illustrating the true reduction of the subluxation resulting from open division of the quadriceps tendon. The angling of the tibial diaphysis results from the abnormal traction through the shortened quadriceps muscle.

DISCUSSION

The deformity of congenital genu recurvatum, though frequently, as in the case just described, encountered as a solitary malformation, is nevertheless not uncommon as a part of the form of generalized contractures of joints which has been variously known as 'arthrogryposis multiplex congenita', 'amyoplasia congenita', or 'myodysplasia congenita', and to which I have applied the name 'myodystrophia deformans foetalis'. In a previous paper⁶⁴ I was able to show that the contractures of this deformity were due to an intra-uterine fibro-fatty degeneration of the striated muscle of the limbs. The naked-eye and microscopic changes in the shortened quadriceps muscle of this case of congenital genu recurvatum demonstrate beyond doubt the underlying similarity of the pathological process in such contractures, whether the condition be localized to one or both quadriceps or whether it be a part only of a generalized deformity associated with club-feet, club-hands, congenital dislocation at the hip-joints, and bizarre contractures of other joints.

In the previous paper referred to above I have been able to describe the

pathological changes in the generalized myodystrophy and to state my opinion that the condition is essentially an intra-uterine degeneration of fully or partially



FIG 479—Section of quadriceps muscle The section shows masses of fibrous tissue with patches of fat ($\times 45$)



FIG 480—Section of quadriceps muscle Areas of normal muscle tissue are seen to be surrounded by an excess of fibrous tissue ($\times 90$)



FIG 481—Section of quadriceps muscle Atrophic muscle fibres and a muscle spindle appear in the lower part of the field, while fat replacement and a nerve trunk are seen in the upper part (> 90)

developed muscle fibres rather than a simple failure of muscle to develop I have also concluded for various reasons that the lesion is a peripheral one and that the

muscular degeneration is not secondary to an embryonic lesion of the nervous system. In this connection it is interesting to note that the degenerated muscles contain their full quota of nerve-trunks and that the presence of many undegenerated muscle spindles, as found in the muscular dystrophies of post-natal life, is a strong argument in favour of a normal sensory supply to the muscle. Finally, Sherrington⁸⁰ has demonstrated by examination of an amyelous foetus that full and normal development of the striated muscles can take place in the complete absence of motor nerve-supply.

SUMMARY

1 A fresh case of congenital genu recurvatum is described along with naked-eye and microscopic observations.

2 A consideration of these findings makes it clear that the deformity is caused by contracture of the quadriceps extensor cruris muscle resulting from intra-uterine fibro-fatty degeneration of the striated muscle fibres.

3 The pathological process is in all respects similar to that described in myodystrophia deformans foetalis.

BIBLIOGRAPHY

- ¹ ARDOUIN, P, *Rev d'Orthop*, 1907, viii, 193
- ² BACILIERI, L, *Arch f orthop u Unfall-Chir*, 1905, iii, 213
- ³ BAKER, H F, *Lancet*, 1881, ii, 951
- ⁴ BALDWIN, C H, *Jour Bone and Joint Surg*, 1926, viii, 822
- ⁵ BARTH, A, *Arch f klin Chir*, 1885, xxxi, 670
- ⁶ BARWELL, R, *Proc Roy Med and Chir Soc*, 1875-80, viii, 218
- ⁷ BAZERT, L, *Rev d'Orthop*, 1925, xii, 155
- ⁸ BERTIN, J, *L'Union med*, 1880, xxx, 616
- ⁹ BISPING, *Zeits f orthop Chir*, 1903, xi, 880
- ¹⁰ BLANC, E, *Gaz med de Paris*, 1884, i, 325
- ¹¹ BOORSTEIN, S W, *Med Jour and Record*, 1926, cxxiv, 541
- ¹² BOORSTEIN, S W, *Amer Jour Child Dis*, 1929, xxxviii, 107
- ¹³ BOULARAN and BOUNHOURE, *Rev d'Orthop*, 1923, x, 245
- ¹⁴ BRODHURST, B E, *On the Nature and Treatment of Clubfoot*, 1856, 32 London
- ¹⁵ CHEYNE, W, *Lancet*, 1890, ii, 925
- ¹⁶ CONRAD, *Corr-Blatt, f schweiz Aertze*, 1875, v, 153
- ¹⁷ CORNET, J, *Arch franco-belges de Chir*, 1927, xxx, 536
- ¹⁸ CURTILLET, J, and LOMBARD, P, *Rev d'Orthop*, 1912, iii, 289
- ¹⁹ DELANGLADE, E, *Ibid*, 1903, iv, 193
- ²⁰ DEROCQUE, P, *Ibid*, 54
- ²¹ DOWD, C N, *Ann of Surg*, 1899, xxix, 345
- ²² DREHMANN, G, *Zeits f orthop Chir*, 1900, vii, 459
- ²³ FRANCINI, M, *Chir d Org di Movimento*, 1917, i, 268
- ²⁴ FRASER, J, *Surgery of Childhood*, 1926, ii, 1105 London
- ²⁵ GIBNEY, V P, *Ann of Surg*, 1899, xxix, 346
- ²⁶ GODLEE, R, *Lancet*, 1877, i, 316
- ²⁷ GRISWOLD, A S, *Jour Bone and Joint Surg*, 1927, ix, 628
- ²⁸ GROSSMANN, J, *Med Jour and Record*, 1927, cxxv, 189
- ²⁹ GUENIOT, *Bull et Mem Soc de Chir*, 1880, vi, 442
- ³⁰ HABS, *Munch med Woch*, 1905, xii, 556
- ³¹ HAMADA, G, *Brit Med Jour*, 1930, ii, 141
- ³² HAMILTON, *Treatise on Fractures and Dislocations*, 1891, 8th ed, 838 London
- ³³ HENRARD, E, *Monats f Geburtsh u Gynaekol*, 1928, lxxx, 317
- ³⁴ HOLTZMANN, H, *Virchow's Arch*, 1895, cx, 272
- ³⁵ HORVATH M, *Wien med Woch*, 1897, xlvii, 1623
- ³⁶ JAGERIK, *Nederl Tyds v Geneesk*, 1927, ii, 476
- ³⁷ JOACHIMSTAL, *Berl klin Woch*, 1889, 923

- ³⁸ KAREWSKI, *Arch f Kinderheilk*, 1891, xii, 234
- ³⁹ KIRMISSON, E, *Rev d'Orthop*, 1903, iv, 413
- ⁴⁰ KNAUER, E, *Monats f Geburtsh u Gynakol*, 1897, v, 1
- ⁴¹ KOFMANN, S, *Arch f orthop u Unfall-Chir*, 1907, vi, 41
- ⁴² KOFMANN, S, *Four Bone and Joint Surg*, 1930, xii, 871
- ⁴³ KONIG, *Berl klin Woch*, 1898, xxxv, 496
- ⁴⁴ KUH, *Deut med Woch*, 1910, xxxvi, 1783
- ⁴⁵ KUSTNER, O, *Arch f klin Chir*, 1880, xlv, 601
- ⁴⁶ LIPSCOMB, T W, *Austral Med Gaz*, 1903, xxi, 568
- ⁴⁷ LITTLE, W J, *On the Nature and Treatment of the Deformities of the Human Frame*, 1853, 319 London
- ⁴⁸ MAAS, H, *Arch f klin Chir*, 1874, xvii, 492
- ⁴⁹ MCFARLAND, B L, *Four Bone and Joint Surg*, 1929, xi, 281
- ⁵⁰ MCGILLICUDDY, T J, *Four Amer Med Assoc*, 1892, vii, 106
- ⁵¹ MAGNUS, F, *Deut Zeits f Chir*, 1905, lxxviii, 555
- ⁵² MASON, *Med Record*, 1877, vii, 42
- ⁵³ MAYER, L, *Amer Jour Orthop Surg*, 1912-13, v, 411
- ⁵⁴ MIDDLETON, D STEWART, *Edin Med Jour*, 1934, xli, 401
- ⁵⁵ MOUCHET, A, *Bull et Mem Soc anat de Paris*, 1904, lxxix, 856
- ⁵⁶ MUSKAT, G, *Arch f klin Chir*, 1897, liv, 852
- ⁵⁷ MUTEL, *Rev d'Orthop*, 1911, ii, 302
- ⁵⁸ MYERS, T H, *New York Med Jour*, 1890, li, 444
- ⁵⁹ OLIVIERAS, D, and BONREPAUX, J, *Rev méd de Barcelona*, 1924, i, 28
- ⁶⁰ OWEN, E, *Lancet*, 1891, i, 989
- ⁶¹ PÉRIER, E, *Bull et Mem Soc de Chir*, 1880, vi, 682
- ⁶² PHOCAS, G, *Rev d'Orthop*, 1891, ii, 50
- ⁶³ POST, G E, *Med Record*, xii, 408
- ⁶⁴ POTEL, G, "Etude sur les Malformations congenitales du Genou", *These de Lille*, 1897
- ⁶⁵ POTEL, G, *Echo Med du Nord*, 1897, i, 372
- ⁶⁶ POTEL, G, *Traite pratique d'Orthopedie*, 1925, 186 Paris
- ⁶⁷ PRINCE, L D, *Surg Gynecol and Obst*, 1917, xxiv, 714
- ⁶⁸ REINER, M, *Zeits f orthop Chir*, 1904, xii, 442
- ⁶⁹ RICHARDSON, W L, and PORTER, C B, *Boston Med and Surg Jour*, 1875, xcii, 321
- ⁷⁰ RIDLON, J, *Trans Amer Orthop Assoc*, 1896, iv, 179
- ⁷¹ ROBERTS, J B, *Ann of Surg*, 1901, xxxiv, 281
- ⁷² ROBERTS, J B, *Ibid*, 1909, xlix, 276
- ⁷³ ROBERTSON, J K, *Glasgow Med Jour*, 1884, xxii, 118
- ⁷⁴ ROBIN, *Rev d'Orthop*, 1914-17, v, 237
- ⁷⁵ ROSENFELD, L, *Zeits f orthop Chir*, 1902, x, 405
- ⁷⁶ SANDERS, A, *Monats f Geburtsh u Gynakol*, 1928, lxxix, 68
- ⁷⁷ SAYRE, L A, *Rev mens d Mal d l'Enf*, 1890, viii, 453
- ⁷⁸ SELLS, H, *Brit Med Jour*, 1883, i, 766
- ⁷⁹ SHATTOCK, S G, *Trans Pathol Soc Lond*, 1891, xli, 280
- ⁸⁰ SHERRINGTON, C S, *Four of Physiol*, 1894-5, xvii, 211
- ⁸¹ SIMPSON, G, *Brit Med Jour*, 1893, ii, 1099
- ⁸² SKELTON, E W, *Long Island Med Jour*, 1910, iv, 24
- ⁸³ SORREL, E, and MME LE GRAND-LAMBLING, *Bull Soc Ped*, 1932, xxi, 169
- ⁸⁴ SPEIRS, H W, *Four Bone and Joint Surg*, 1927, ix, 469
- ⁸⁵ STEELE, A J, *Trans Amer Orthop Assoc*, 1896, ix, 175
- ⁸⁶ TARNIER, *Bull et Mem Soc anat de Paris*, 1854, 109
- ⁸⁷ TAYLOR, H L, *Trans Amer Orthop Assoc*, 1895, viii, 280
- ⁸⁸ TIMMER, H, *Nederl Tyds v Geneesk*, 1892, xxviii, 778
- ⁸⁹ TRIDON, P, *Rev d'Orthop*, 1905, vi, 497
- ⁹⁰ TSCHARNOMSKAJA, J J, *Zentralb f Chir*, 1902, xxix, 791 (Abstract)
- ⁹¹ VON SALIS, H, *Deut Zeits f Chir*, 1908, xciv, 149
- ⁹² WEHSARG, R, *Arch f orthop u Unfall-Chir*, 1905, iii, 197
- ⁹³ WENTWORTH, E T, *Four Bone and Joint Surg*, 1928, x, 585
- ⁹⁴ WITTEK, A, *Deut Zeits f Chir*, 1930, ccxxv, 308
- ⁹⁵ WOLFF, J, *Zeits f orthop Chir*, 1893, ii, 23
- ⁹⁶ WUTZER, *Arch f Anat u Phys (Muller)*, Berlin, 1835, 385

OBSERVATIONS ON FISTULA IN ANO IN RELATION TO PERIANAL INTRAMUSCULAR GLANDS WITH REPORTS ON THREE CASES

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It was pointed out as far back as 1880 by Herrmann and Desfosses¹ that some cases of fistula in ano might be caused by glands penetrating deeply from the lower rectal mucous membrane into the perianal tissues. "Au point de vue chirurgical ces longs conduits tortueux coiffes a leur extremite par de petits follicules clos, presentent un certain interêt, en regard notamment au rôle qu'ils peuvent jouer dans la production des fistules compliquees qu'on rencontre si frequemment dans cette region." The same authors showed that the glands reached and actually invaded the muscle of the internal sphincter after passing through the muscularis mucosæ.

In 1929 Lockhart-Mummery, in an address on fistula in ano at the Royal Society of Medicine,² referred to Cuthbert Dukes's statement that intramuscular glands can often be seen in complete sections of the anal region, and to his suggestion that they might be a possible cause of fistulæ. He pointed out that these glands had, until comparatively recently, been apparently overlooked by anatomists and surgeons.

ANATOMY AND DEVELOPMENT OF PERIANAL INTRAMUSCULAR GLANDS

These glands are variable in number and distribution, and seem to serve no particular purpose. It has been suggested that they are the remains of odoriferous glands, which may have played an important rôle in sex attraction before our ancestors assumed the erect posture. *Fig 482* illustrates these glands and their relation to the anal canal.

Further interesting details explaining the development of these glands were given by Professor H. A. Harris³ when he spoke on "Some Embryological Aspects of the Problem Involved" in the above discussion on fistula in ano. Harris pointed out that when parent tubes give rise to daughter tubes—such as the œsophagus to the trachea and to the bronchial tree, the duodenum to the pancreas, to the liver, and to the biliary system, and the rectum to the urogenital sinus—then irregularities in the muscular and epithelial layers of the original tubes are not infrequent. Atresia, stenosis, and cyst formation may occur, whilst the muscular layer, which is weakened by the outgrowth and retarded in development, lends itself to diverticulosis.

Harris also emphasized that certain of the intramuscular glands grow deeply to reach the inner circular layer before the muscularis mucosæ is differentiated.

Histologically the glands are lined by transitional epithelium from the epithelium of the anal canal at the anorectal junction, and the structure is that of

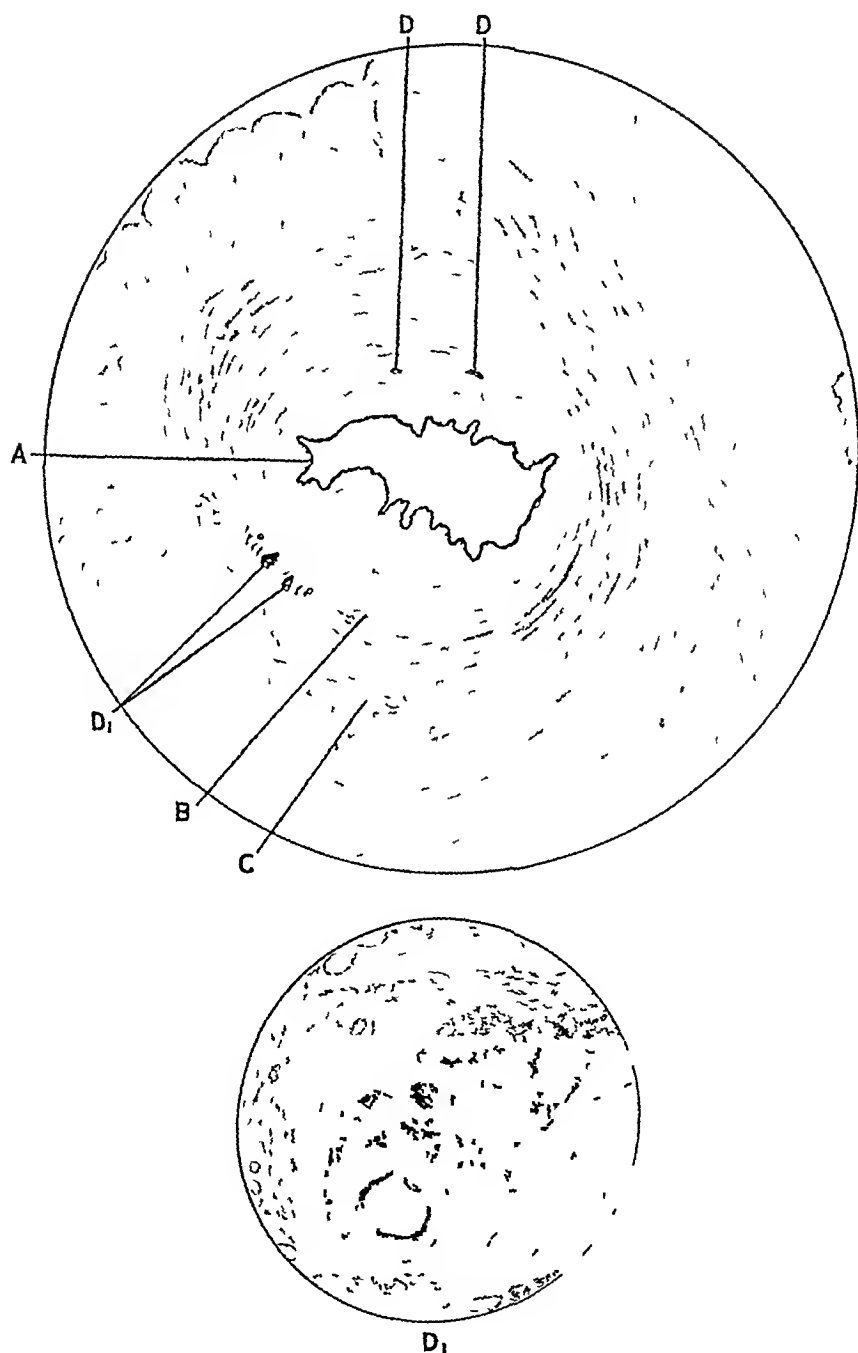


FIG 482.—Transverse section of the anal canal. A, Stratified epithelial lining. B, Internal sphincter. C, Tendon of longitudinal muscle of rectum surrounded by the external sphincter. D, Intramuscular glands buried in the substance of the internal sphincter muscle. D₁ (inset), Higher magnification of an intramuscular gland surrounded by round-celled infiltration. (From St Mark's Hospital Museum.)

either convoluted mucous or of sweat glands. The glands grow outwards into the loose tissue within the internal sphincter and into the limiting annulus of connective tissue which separates the internal sphincter above from the external sphincter below. Before the muscularis mucosæ is developed the glands may arrive at and actually penetrate the internal sphincter and the external longitudinal coat, and even spread. (1) To the superficial surface of the levator ani and so into the ischiorectal fossa, (2) Deep to the levator ani into the true pelvis, (3) Into the substance of the levator ani muscle.

In post-natal life these glands may be recognized deep to the internal sphincter, in the substance of the external sphincter, or even in the levator ani.³

4. THE RELATIONSHIP OF ANORECTAL GLANDS TO FISTULA IN ANO

Having established the presence, development, and position of deep intramuscular perianal glands, it is not difficult to appreciate that pathological changes may readily occur in them.

It seems probable that these glands are, for the most part, vestigial and often lose connection with the anal canal by obliteration of the ducts during development and that in some instances when the duct is patent infection from the anal canal takes place, as is illustrated by the three cases recorded below. In other instances infection may occur in the glandular substance when there is no direct communication with the anal canal, or when the orifices of the ducts have become blocked by congestion of the mucosa and submucosa.

If an abscess occurs in one of these glands, it is easy to visualize the onset of a perianal or ischiorectal abscess and a subsequent fistula. On the rarer occasions when one of these glands has penetrated to the deep surface of the levator ani, we may have an explanation for the development of a pelvirectal abscess and fistula.

The presence of these deep glands emphasizes the importance of a careful search for a rectal opening when dealing with a supposed blind external fistula. When such a track is overlooked, permanent relief cannot be effected, and the fistula will not be cured.

In the past it has been a little difficult to explain why it happens that so many fistulæ in ano appear to have been chronic from the first and have failed to present any initial history of an acute onset or of abscess formation. It now seems quite easy to believe that a chronic origin may well be the truth in some cases, should a mild infection of a perianal gland occur and the existence of a patent duct permit drainage into the anal canal. In other instances in the absence of a patent duct it is equally easy to believe that infection of the perianal glands may well be responsible for many of the acute or subacute abscesses which arise for no apparent reason.

Unless tuberculous disease or malignant invasion of a chronic fistula is suspected, it is not the usual practice to excise fistulous tracks, and submit them to histological examination, so that it is, perhaps, not surprising that the observations recorded below appear to have escaped recognition in the past.

The existence of these perianal glands, often with patent ducts leading into the rectum, the tuberculous origin of many fistulæ, and the fact that malignant

changes sometimes occur in fistulous tracks combine to emphasize the importance of a histological examination when operating for fistula in ano

Obviously it can happen only occasionally, and then in the case of a recently formed abscess or fistula, that the epithelium of these glands and their ducts lining either abscess or fistula is recognized histologically. Once such a case has become chronic, granulation tissue will have replaced the epithelium and the real origin becomes obscured.

Although the theory that these deep perianal glands might cause an abscess or fistula was tentatively suggested as far back as 1880, and has been revived more recently, no clinical and histological evidence appears to have been brought forward to support this view until one of us (C. G.-W.) in January, 1932, before the Subsection of Proctology at the Royal Society of Medicine, recorded a case which seemed quite clearly to support the above views (*Case 1* below). Since then two other similar cases have been met with and are now reported. These cases with histological illustration are, in all probability, the first to be recorded, at any rate in this country. It is hoped that the records will stimulate others to confirm the relationship between some of the fistulae in ano and the intramuscular glands.

CASE REPORTS

Case 1—An Oxford undergraduate, aged 21, came under the care of one of us (C. G.-W.) on Dec. 9, 1931, complaining of pain in the region of the anus for the past three days.

ON EXAMINATION—There was a small localized inflammatory swelling about ½ in. from the anal margin on the right side, which was recognized as an early ischio-rectal abscess.

OPERATION—At operation the anal canal was examined for evidence of any communication between the abscess and the anal canal. On squeezing the abscess area a little pus exuded from beneath one of the valves of Morgagni. A director was passed through this opening into the abscess cavity and the abscess incised through the skin. The track leading from the abscess to the anal canal passed through the substance of the external sphincter. The lining of this track was carefully dissected out and the radical operation completed (*Fig 483*).

PATHOLOGICAL REPORT—The following is a copy of the report made by Dr. Cuthbert Dukes on the specimen sent to him. The fistulous track has a complete lining of a transitional type of epithelium, mostly from three to six cells in depth. The subepithelial tissues are infiltrated with inflammatory cells, chiefly small and large mononuclear cells. There are also some collections of inflammatory cells in the muscle and connective tissue surrounding the fistula, but the histology is not in any way suggestive of a tuberculous lesion.

Comment by Dr. Dukes—Fistulous tracks are rarely lined with epithelium. The congenital sacrococcygeal fistula is lined by stratified epithelium with rudimentary sebaceous glands and hair follicles, none of which are found in this case. A long-standing septic fistula may have a well-developed lining of stratified epithelium due to the skin growing in from the edge, but the epithelium lining this fistula has not these histological appearances. The track of this fistula closely resembles a duct of one of the intramuscular glands which pass from the upper part of the anal canal into the sphincter muscles.

Case 2—A man (C. E. D.), aged 48, was under the care of one of us (H. D.) in May, 1932. In January, 1932, the patient noticed a painless swelling to the right of the anal margin. By February the swelling had spread, was painful, and after fomentations burst and discharged a small amount of pus, which continued until he was first seen.

On May 23 he was admitted to hospital and a small external fistula was found about 1½ in. from the anus at 11 o'clock. No induration was detected between the rectum and the fistula, but pressure on the area exuded a small bead of thin pus.

OPERATION (May 26)—Under low spinal anæsthesia the fistula was laid open on a director and a granulating surface ending in a small abscess cavity was revealed. The area involved, together with the surrounding skin, was excised *en masse*. On cleaning the wound a thin bead of pus exuded from the centre of its soft fatty surface. This area was explored



FIG. 483.—Case 1. Illustrating a fistula arising from an intramuscular gland. A, Section of the track of an acute ischio-rectal abscess passing through the sphincter into the anal canal. The track is lined with epithelium similar to that of the intramuscular glands ($\times 16$). B, Higher magnification ($\times 100$).

with a fine probe and a track found leading through the lower fibres of the external sphincter into the rectum. The track was excised.

After operation the excised pieces were threaded on silkworm gut, and the epithelial-lined track could be seen running directly into the abscess cavity (Fig. 484). After preservation in formalin the duct showed up even more clearly.

PATHOLOGICAL REPORT—Dr Cuthbert Dukes reports on the excised pieces as follows—

1 Region of External Opening—In this region the fistulous track is lined with granulation tissue of the type found in infections with pyogenic bacteria and there is no sign of an epithelial lining

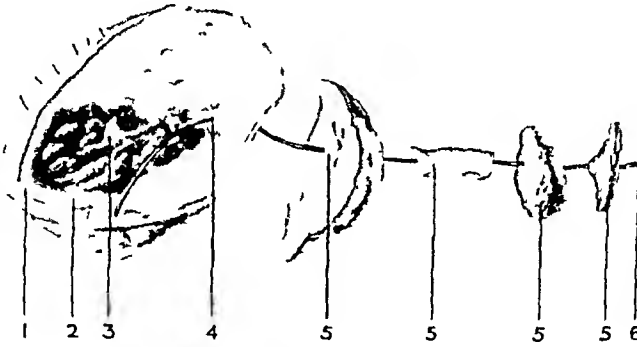


FIG 484—Case 2 The fistulous track dissected out to the rectum 1, The external orifice of the fistula, 2, The skin of the buttock surrounding 1, 3, The granulating base of the fistulous track, 4, The orifice of the epithelial-lined track in 3, 5, The track running through to the rectum, 6, A piece of silk-worm gut

2 Deeper Portion of Tract—In this region the fistula is lined with a complete layer of stratified epithelium containing neither sebaceous nor sweat glands nor hair follicles. Interspersed in the surrounding tissues are a few collections of inflammatory cells with some giant cells amongst them. Another piece of tissue from the same region includes only a portion of the fistula, but shows the same features.

I think a possible explanation is that this fistula arose from one of the intramuscular glands which open into the lower part of the anal canal—that is to say, below the level of the valves.

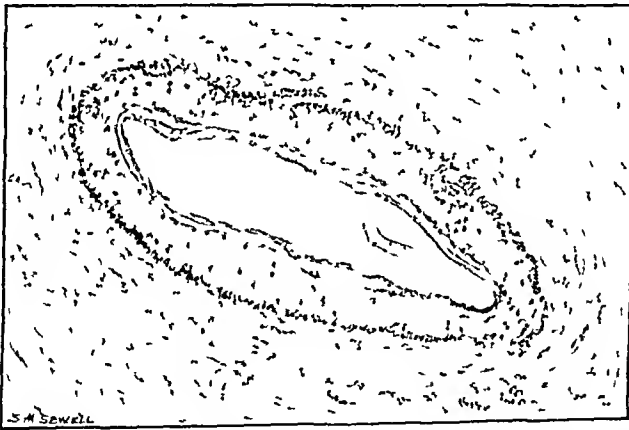


FIG 485—Case 2 Microscopic section of the epithelialized track ($\times 40$)

This portion of the anal canal is covered with stratified epithelium not containing any sweat glands or sebaceous glands or hair follicles. The track of the fistula has a similar lining (Fig 485). The fact that there is no evidence of any epithelium at the external opening proves that this is not a simple case of epithelialization of the track of a chronic fistula.

Case 3—A man (C G), aged 36, under the care of one of us (C G-W) was admitted to hospital with the following history Six months previously he began to be troubled with piles Four months previously he first noticed some yellowish discharge from the anus Recently there has been some pain and tenderness at the anal margin

ON EXAMINATION—On rectal examination a small perianal swelling was detected at the anal margin anteriorly, about the size of a pea The swelling was firm and appeared to be in the substance of the sphincter On examination of the anal canal a small fistulous track, about $\frac{1}{2}$ in long, was detected leading directly into the swelling

OPERATION (June 18, 1934)—A probe was passed along the track into the abscess cavity and the abscess cavity dissected out intact from the substance of the external sphincter, and then a radical operation for direct fistula was carried out Some hæmorrhoids were also ligatured Recovery was straightforward

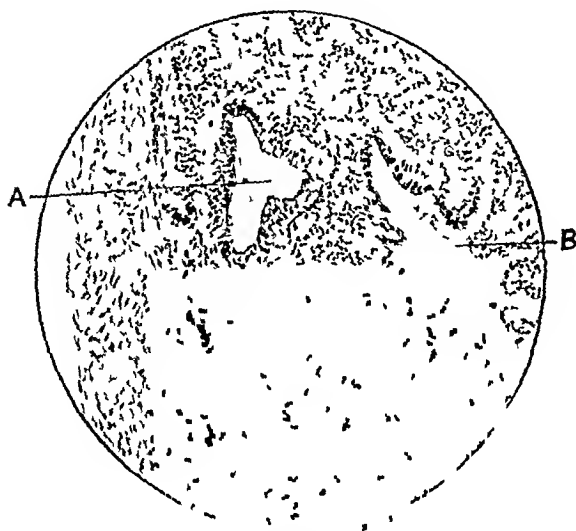


FIG 486—*Case 3* Abscess surrounding intramuscular gland The section passes transversely across two terminal branches of an intramuscular gland In the branch marked A the epithelial lining is intact, but in B only the upper half of the lumen is covered by epithelium, and the appearances suggest that the duct is opening directly into the abscess ($\times 70$)

PATHOLOGICAL REPORT—Dr Cuthbert Dukes reported as follows—

Tissue from Anal Region—The fragment consists of sphincter muscle and connective tissue from the anal region A small abscess is situated between the fibres of the sphincter muscle, and in contact with this is the duct of a gland lined by transitional epithelium This duct has the histology of an intramuscular gland arising from the anal canal, and the appearances suggest that the abscess is related to the presence of the gland (*Fig 486*)

SUMMARY

- 1 Some observations are made on deep intramuscular perianal glands in relation to the cause of abscess and fistula in ano
- 2 Three cases of fistula in ano lined with glandular epithelium are recorded
- 3 Microphotographs of each case are submitted

REFERENCES

- ¹ HERRMANN, G, and DESFOSSÉS, L, *Comptes rend Acad des Sci*, 1880, xc, 131
- ² LOCKHART-MUMMERY, J P, *Proc Roy Soc Med*, 1929, xxii, 1331
- ³ HARRIS, H A, *Ibid*, 1341

SCHIMMELBUSCH'S DISEASE OF THE BREAST AND DR. A LACASSAGNE'S EXPERIMENTS ON MICE

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THE development of carcinoma in the breasts of young male mice after the injection of œstrin is one of the most interesting experiments Dr Lacassagne has recently performed. I am very grateful to him for having shown me his microscopical sections and for his courtesy in allowing me to reproduce them before he has published them himself. The object in writing this article is to point out that the morphological appearances seen in these sections are the same as those in all the stages of the process that I have described as Schimmelbusch's disease when it ends in carcinoma. The similarity of my description of this disease and the experimental changes that occur in the breasts of his mice had already struck Dr Lacassagne before I had seen his sections and discussed the matter with him.

My conception of Schimmelbusch's disease differs from Schimmelbusch's description only in one respect. To his description I add the formation of cysts as an essential preliminary stage to the procession of events that may end in carcinoma. Otherwise, with minor exceptions, I agree entirely with his masterly treatise, and see no reason why his name should be omitted from the nomenclature of the disease he described.

With my addition, Schimmelbusch's disease begins in a desquamative epithelial hyperplasia, sometimes in the ducts only, and sometimes in ducts and their acini. The disease may affect one duct only, or only one duct and its acini. It may be more widely distributed. The affected ducts are dilated by the presence of colostrum-like cells in a fluid medium. The end of this stage is the formation of duct and acinous cysts. It begins in the late twenties or early thirties, and may last an indefinite time or pass on to its next stage at once. It is interesting to quote at this point an observation Dr Lacassagne has made on the contents of these cysts. He says: "*Enfin nous avons commence a rechercher l'hormone dans le tissu mammaire de seins preleves chirurgicalement. Dans le cas d'une Femme de 57 ans un epithelioma occupait une portion de la glande, des sections profondes, dans la partie non cancéreuse de l'organe, ont permis de faire sourdre un liquide provenant de l'ouverture de petits kystes. Ce liquide, injecte a la dose de 1 cc, a provoque l'œstrus. En revanche, un broyat du tissu cancéreux lui-même s'est montré inactif.*" And in the paper in which he made this statement he concludes: "*Le colostrum contient normalement une quantité certainement très élevée de folliculine. Cette hormone peut également être trouvée dans la glande mammaire de Femmes atteintes de cancer du sein.*"

If the process proceeds at once, the next stage—which really is the beginning of Schimmelbusch's description—is the development of multiple epithelial neoplastic growths within the cysts. These tumours do not transgress their dilated normal

boundaries. They may or may not be papillomatous in character, although they very often are. They may be limited only to the ducts, or they may also grow in the acini. I have not seen them only in the acini.

This second stage usually occurs in the late thirties or early forties. The tumours may remain within their normal boundaries for an indefinite time, although they continue to grow in size and increase in number. If the process continues, the next and final stage of carcinoma begins in the late forties and early fifties. The time of appearance of these three stages is not always so stable as their sequence. For example, Mr Geoffrey Keynes has recently allowed me to cut whole microscopical sections of a breast he removed from a woman aged 27 years. According to my conception of them, the disease had already passed through the cystic and benign neoplastic stages, and reached the carcinomatous end—all of which stages are to be seen in the sections.



FIG 487 —Photograph of the normal and immature breast of a male mouse. A, Skin, B₁ and B₂, Two ducts, C, Muscle fibres

Turning now to Dr Lacassagne's experiments on young male mice. He injected œstrin into their bodies and cut microscopical sections of their breasts in consecutive stages of their treatment. He used mice belonging to carcinoma strains and also mice in which no such strain was known to exist. In the breasts of the latter carcinoma developed later than in those of the former. Dr Lacassagne states that the adeno-carcinomata following the administration of œstrin behave like the disease that arises spontaneously in mice. Metastases occur with great frequency and generally in the lungs. At this point the reader should study the photographs of Dr Lacassagne's sections (*Figs 487-493*).

After studying these sections the reader will be struck by several sets of impressions, to some of which I now draw attention. It will be fairly obvious that the changes in the newly-formed breast tissue are the same in kind and sequence as in Schimmelbusch's disease. It also appears obvious that the changes leading

to carcinoma do not occur in the normal immature breasts of the mice, but that they appear in the new and abnormal breast tissue. Considering the size of the animals, the cysts are large ones. They resemble the duct cysts of Schimmelbusch's disease in that they do not coalesce to form larger ones. The sections support my contention that Schimmelbusch's disease is a disease *sui generis* and unrelated to

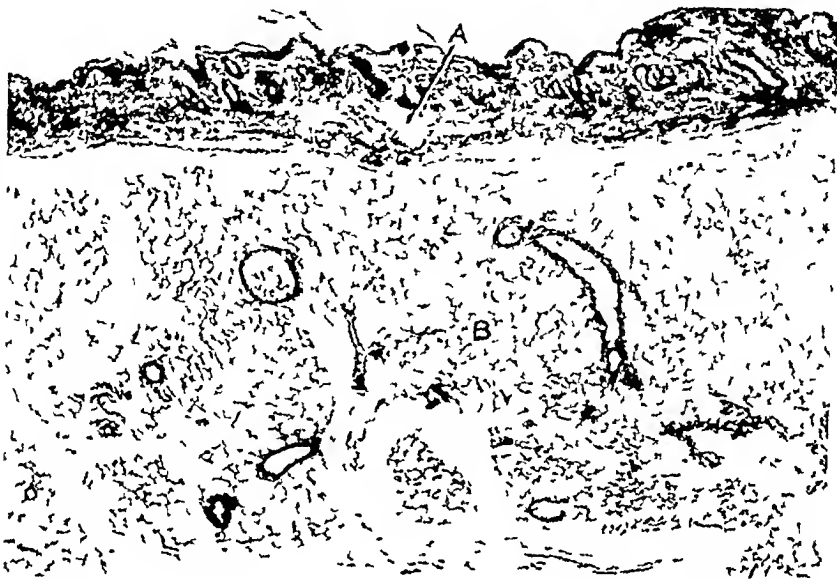


FIG 488—Photograph of the breast of a male mouse after forty-eight days' treatment with æstrin. A, Skin. B, A layer of fat in which can be seen five or six ducts. There is a slight increase of breast tissue, the dilated ducts of which contain desquamated epithelial cells.

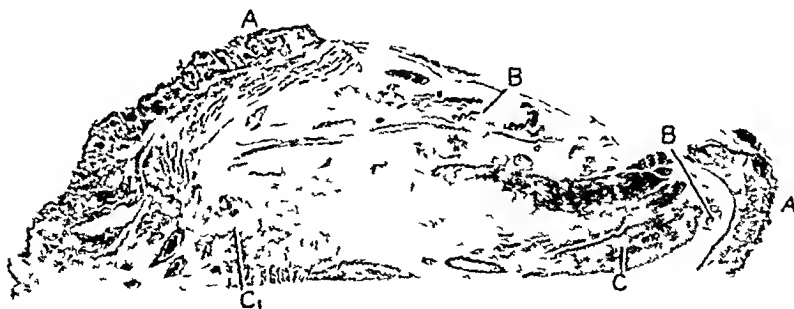


FIG 489—Low-power photograph of a whole section of the breast of a male mouse after eighty-five days' treatment with æstrin. A, Skin. B, Breast tissue that shows a decided increase in amount and a state of multiple cysts, most of which appear to be formed by dilated ducts. C and C1, Musculature (Cf Fig 490).

mazoplasia (so-called chronic mastitis), of which there is not the slightest sign. They also support my belief that all the cystic breasts in Schimmelbusch's disease should be removed. In carrying out this practice for some years I have met with the following results in the total excision of these breasts: (1) Some of them have been only cystic, (2) Others contained cysts with papillomatous and other growths

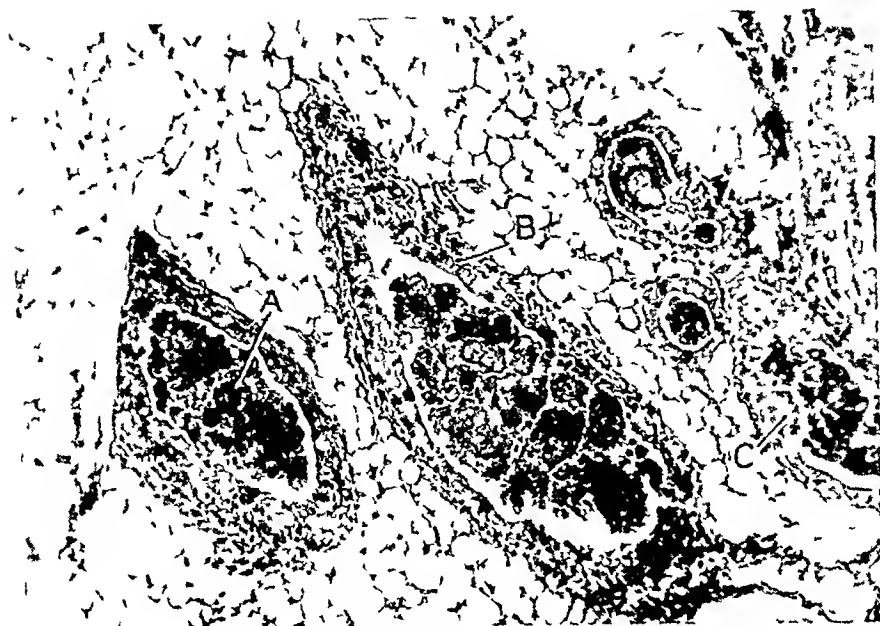


FIG 490—High-power photograph of breast tissue from Fig 489 (eighty-five days). Epithelial neoplastic cells (among which many mitoses can be seen) have formed round the margins of what are apparently ducts (A, B, C).

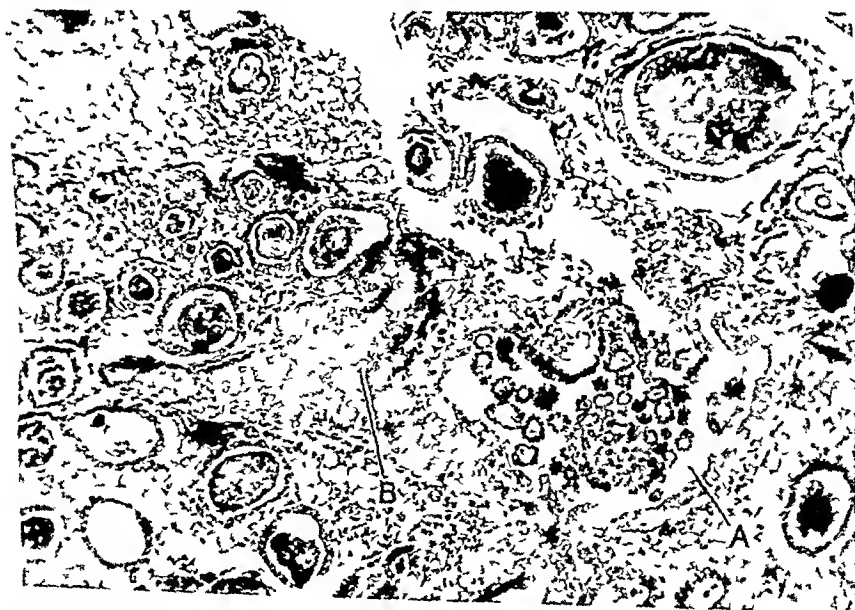


FIG 491—Photograph of a part of the breast of a male mouse 143 days after treatment with oestrin. There has been an enormous increase of breast tissue, which is in a multiple cystic state. At A is apparently a duct in which epithelial neoplastic cells (B) have developed and apparently are still contained within their normal but dilated boundary. There are many mitoses visible in the epithelial neoplasia.



FIG 492—Photograph of part of the breast of a male mouse 179 days after treatment with œstrin. The section shows the enormous increase of breast tissue as in the breast of Fig. 491, also its multiple cystic state. At A is a well-developed carcinoma (Cf Fig. 493)

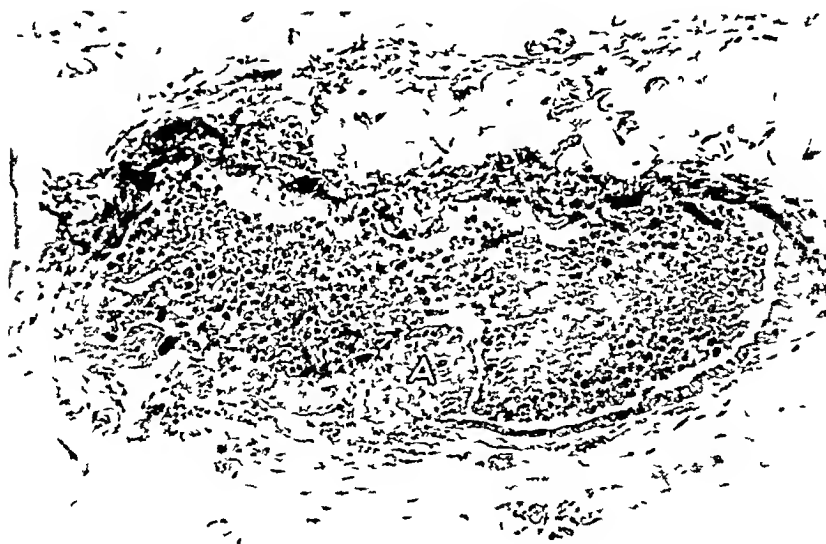


FIG 493—Photograph of apparently a duct from a part of Fig. 492 far remote from the area of carcinoma. Epithelial neoplasia (A) has occurred that is still confined within its normal but dilated boundary

confined within their normal boundaries, (3) Others contained definite and unsuspected early carcinoma, and (4) Finally I have met with other instances from which a cyst or cysts had been very locally excised and had subsequently developed carcinoma

Unfortunately one man's experience is insufficient to provide enough material to form an impressive notion of the difference in numerical relationship between these four conditions. My own experience is that the conditions (1) and (2) are more frequent than (3) and (4), my impression being that (2) is more frequent than (1). Conclusions upon this matter are of no value whatever when they are based only on a partial examination. The only valuable evidence is that based upon the examination of microscopical sections made of the whole mammary glands

The remaining practical issue of these experiments is as follows —

Are patients suffering from mazoplasia running any risk by the administration of 'ovarian residue', i.e., ovarian extract deprived of its luteal contents? It would be a pity if its use in this condition were contra-indicated, because it so often relieves the symptoms. I cannot conceive that it is dangerous. In considering this point it must be realized that every pregnant woman and mare is flooded with œstrin with no bad effect. It may well be that the œstrin may make matters worse when carcinoma exists in the breast of a pregnant woman, and may be the cause of the particularly malignant type of the disease which is popularly supposed to exist when it occurs in pregnancy.

In all my sections I cannot discover an indication that mazoplasia is related to carcinoma. These sections seem to me to show that it would be unwise to administer 'ovarian residue' in women suffering from cystic breasts.

BIBLIOGRAPHY

- LACASSAGNE, A., "A Propos d'une Pathogenie de l'Adeno-carcinome mammaire Recherche de la Folliculine dans le Colostrum", *Comptes rend Soc de Biol*, 1934, cxvi, June, 844
- LACASSAGNE, A., "Sur la Pathogenie de l'Adeno-carcinome mammaire de la Souris", *Ibid*, cxv, March 3, 937
- LACASSAGNE, A., "Influence d'un Facteur familial sur la Production, par la Folliculine, de Cancers mammaires chez la Souris male", *Ibid*, 1933, cxiv, Oct 21, 427
- LACASSAGNE, A., "Apparition de Cancers de la Mamelle chez la Souris mâle, soumise a des Injections de Folliculine", *Comptes rend Acad des Sci*, 1932, cxcv, Oct 10, 630
- SCHIMMELBUSCH, C., *Arch f klin Chir*, 1892, xlv, 117
- CHEATLE, G. L., and CUTLER, MAX, *Tumours of the Breast*, 1931, 92-159
- RECLUS, P., *Bull et Mem Soc anat de Paris*, 1883, lxxviii, 428
- WHEELER, SIR W. I. DE C., "Breast Tumours", *Med Annual*, 1931, 79
- GOORMAGHTIGH, N., and AMERLINCK, A., "Realisation experimentale de la Maladie de Reclus de la Mamelle chez la Souris", *Bull Assoc fran pour l'Etude du Cancer*, 1930, xiv, No 7, July

OSTEO-ARTHRITIS OF THE HIP-JOINT

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THE condition usually described as osteo-arthritis of the hip-joint is essentially a disease of middle and later life. The very greatest ingenuity has been shown in its classification, and each new investigator feels the urge for further subdivision, until in many of these classifications it is almost impossible to recognize what is after all a very common condition. Fortunately the classification which is generally accepted at present is probably the simplest of all. In it the condition is divided into two main groups (1) The infective or toxic, and (2) The traumatic. From the fact that two such widely differing causes may produce conditions in the joint which are apparently similar it would seem that osteo-arthritis of the hip is not

a definite disease of itself, but is rather the reaction of the joint and articular cartilage to adverse conditions which lead to a loss of their vitality, or to an alteration of the line of weight-bearing through the joint surfaces and must cause an abnormally increased pressure over a restricted area.

Age Incidence—A study of 89 cases of osteo-arthritis of the hip, both unilateral and bilateral, on which this paper has been based, reveals some very interesting points in regard to the age of onset of the first symptoms of trouble. In the bilateral cases the average age of onset was 53 years, while in the unilateral group it was 34 years. In calculating these two figures it was found that in a large proportion of the unilateral cases there was a history of some injury or strain of the hip in adolescence which gave rise to trouble for a limited period, and then apparently recovered.



FIG 494—A case of osteo arthritis of hip the result of Perthes disease in early childhood

In these cases the time of onset was reckoned from this early injury and not from the occurrence of stiffness and pain at a later age. It has been possible to find the old X-ray photographs (Fig 494) or full notes of these injuries in only five cases, three of these show typical X-ray changes of osteo-chondritis

deformans juvenilis, or Perthes' disease, and in the other two there are distinct signs of a partial slipping of the neck of the femur on the epiphysis. In all five of these cases there has been an alteration in the normal joint relationship between the head of the femur and the acetabulum.

Normally the head of the femur is so shaped in relation to the socket that almost half of its articular surface is in contact with the acetabulum at any angle, but if this relationship has been altered by the widening and irregularity of the head of the femur, or by the sliding upwards of the neck, then more pressure will be borne on a restricted area, and alterations of the articular surfaces of the femur and acetabulum are the natural consequence. That pressure applied obliquely to a normal joint, or with an uneven distribution, may result in osteo-arthritis is well shown by the reaction of the ankle-joint to the strain cast on it by a malunited fracture of the leg, or, as instanced by Arbuthnot Lane, in the unilateral osteo-arthritis of the lumbar spine resulting from the constant carrying of heavy weights on one shoulder by dock labourers. There seems no doubt also as to the possibility of the occurrence of osteo-arthritis of the hip-joint resulting from one severe trauma, but strangely enough in very few instances does the patient associate his disability with one injury, however severe.

X-ray Appearances—The X-ray appearances are usually typical, there is the loss of joint space due to the wearing away of the articular cartilages, atrophy of the bones due to a diminished lime content, and around the articular margins of the acetabulum and femur lies a rim of osteophytic outgrowths. In bilateral cases this is the X-ray finding with the head of the femur normal in shape and position, or approximately so, but in unilateral cases there is usually in addition some distinct alteration in the shape of the head of the femur. As a rule this is flattened and depressed with its convex surface forming a continuous curve with the upper surface of the neck of the bone, and the great trochanter is raised above its normal site as a result of some old-standing injury or deformity of the head or neck of the femur.

Etiology.—It seems clear then that in osteo-arthritis of the hip-joint we are dealing with two separate and distinct problems. In the toxic, or infective, arthritis there is the problem of a generalized infection which has led to widespread joint changes, and the causative infection—of whatever type—is probably still present. In osteo-arthritis of one hip—whether or not there has been any infective process—the exciting cause of the condition has probably been trauma, or an alteration of joint mechanics, so that the fear of spread of the condition to other parts is not present to the same extent. The two types—infective and traumatic—have many points of similarity, the X-ray changes show only minor differences in regard to bony outgrowths. Limitation of movements occurs equally in both conditions, but on account of their essentially different origins there are certain fundamental differences in the treatment which should be adopted for their relief.

It is with the object of clarifying our views as to the best line of treatment to be adopted for various types of osteo-arthritis of the hip-joint that the following review has been made of the treatment and after-results of 89 cases dealt with during the past fifteen years. This number includes unilateral and bilateral cases, in addition to a few in which the arthritis was associated with some type of spondylitis.

TREATMENT

In discussing treatment I do not propose to say much about general treatment, which is usually carried out in a routine manner in all these cases. Sepsis—from whatever source—must be sought for and if found must be dealt with efficiently. Physiotherapy by massage, radiant heat, ionization, mud baths, hydrotherapy, etc., may be tried, either singly or in rotation, but unfortunately they usually result in little or no success. Temporarily at least most of them appear to do good, after having the treatment the patient feels better and agrees that the joint is less tender and more free, but after an hour, or even less, the condition is as before until the next treatment is given. Sometimes if it is carried out in a spa where the patient is away from business worries, is on a diet, and is free from intestinal stasis, there may be a definite diminution in the discomfort felt in the joint, but if bone changes are present no form of non-surgical treatment can give permanent relief, or can produce any lasting improvement.

Our choice of possible surgical procedures may shortly be classified under the following six headings: (1) Manipulation of the joint in order to increase the range of movement, (2) Rest of the joint so as to reduce the inflammation present and diminish the pain, (3) The formation of a new joint at the site of the old one—arthroplasty, (4) The formation of a false joint close to, but not at the original site—pseudarthrosis, (5) Complete destruction of the joint and ankylosis by operative means—arthrodesis, (6) An alteration of the weight-bearing through the joint by means of an osteotomy—bifurcation operation. With such a wide selection of surgical procedures our difficulty usually is not in performing some definite surgical operation, but rather in choosing which of the many possible procedures is the most suitable in any particular case. The following resume has been made in order that the choice may be something better than a matter of chance.

1 Manipulation—When the condition is met with in its early stages the bone changes, as shown in the X-ray photographs, are usually slight. There may, indeed, be only a sharpening of the articular edges, and the patient complains not of a complete loss of movement but rather of an inability to perform some particular movement which he has previously been in the habit of doing regularly, there is often a difficulty in putting on or tying his shoe. Rotary movements, such as swinging at golf, may cause a sudden stabbing pain, and on clinical examination it may be found that only one movement of the joint is in any degree limited. In a case such as this (and in a patient otherwise healthy) gentle manipulation of the joint under an anæsthetic is likely to increase the range of movement and enable the patient to perform the acts which have previously been impossible. There is one very important clinical point to be taken into consideration before such a manipulation is undertaken. If the patient states that the pain caused by attempting the restricted movement is of short duration and disappears after a few minutes' rest, then the result of manipulation is usually satisfactory, but if he states that the pain only disappears after a considerable time, the result of such a manipulation will probably be very disappointing.

The manipulation must not be haphazard but must be carried out in a routine manner, the hip is gently moved through its whole range, each movement being performed once and once only. After-treatment consists in free use of the joint and exercises so designed that the movement which was limited previously is

performed at least three or four times a day. The result in a suitable case is often dramatic, and the patient is apt to praise unduly the manipulator, whether qualified or unqualified, and for a period he contrasts the great benefit he has received from the manipulation with the disappointing results of all previous treatment. The period of relief varies very considerably from a month to five or six years, but in the average case improvement lasts for about a year (Figs 495, 496). If, however, manipulation is carried out in a more advanced case, no improvement is seen—in fact, the patient may only be made more tender and crippled than before.

This form of treatment was carried out in 27 cases of this series, with the following results. In 9 cases the result was a complete failure, as pain and limitation were increased and some other form of treatment had to be adopted



FIG 495—Early osteo-arthritis of the hip, relieved of pain by manipulation



FIG 496—The same patient three years later on, showing that the condition of the hip has progressed. Further manipulation at the patient's own request, for a return of pain, was not successful.

within a few months. In 11 cases there was a definite improvement—pain was less and movements were increased for an average of one year after the manipulation. In the other 7 cases improvement continued for a period between three and five years, but in every case where it has been possible to find the subsequent history there has been a return of the disability with gradually increasing limping of the joint as shown in the X-ray photograph, and in this stage a second manipulation would only increase the patient's discomfort.

2 Fixation and Rest—When the condition is more advanced, with a more general limitation of movement and with definite X-ray changes, then manipulation of the joint—except when used to correct a deformity—cannot be of benefit, and some other form of treatment must be undertaken. Of all the possibilities open to us, by far the simplest is rest and support of the joint. This is commonly carried

out in one of two ways. Firstly—and in my opinion inadvisedly—by the application of a walking caliper which to some extent relieves the hip from body weight. This cannot prevent movements of the joint, and therefore has no effect on the inflammation which is the direct result of the rubbing together of two roughened and irregular articular surfaces. The second method is the application of a plaster spica in which the patient may walk. While this latter does not relieve any of the body weight, it is much more efficient than the former, because movements of the joint are prevented and the inflammation present is thereby reduced.

The latter treatment has been adopted in 24 cases, in 20 of these the condition was a monarticular traumatic arthritis, and in the other 4 a painful infective arthritis. In the case of the 4 latter the result as far as relief from pain was concerned was



FIG 497—X-ray of a case of early osteo-arthritis of the hip, successfully relieved of pain by plaster spica for three months



FIG 498—X-ray of the patient shown in Fig 497 five years later, still free from pain, but the osteo arthritis has progressed

excellent, but while the pain was diminished or lost so also was movement, and ankylosis—complete or partial—was the invariable result. In the other 20 cases the result was good. In every case pain was temporarily diminished, and as a direct result of the rest of the inflamed joint the range of voluntary movement was increased in 6 cases. This increase of movement is of great interest as it illustrates the beneficial effects of the elimination of the constant irritation of the joint which is caused by the rubbing together of the roughened bony surfaces. In none of the cases of monarticular osteo-arthritis was there an appreciable permanent diminution of movement attributable to the prolonged fixation in plaster.

Unfortunately, although the treatment by fixation in plaster gave these excellent results it did not in any way alter the pathological conditions present in the joint (Figs 497, 498), so that a recurrence of inflammation and pain took place

in most cases in a period of three months to eight years. The great advantage of this line of treatment is that there is no operation and no period of recumbency—two factors which are of importance in these patients, some of whom are unsuitable subjects for surgical operations, while others have a strong bias against operations of any type. During the wearing of the plaster spica exercise is permitted, and with the diminution of pain the patient can frequently walk about more freely than before the application, but there are undoubtedly certain disadvantages. Firstly, the application of a plaster spica which will remain in position on the hip of a very fat woman is usually extremely difficult, and secondly the weight of the plaster may be so great that the patient feels quite unable to carry around this extra load.

3 Arthroplasty—Theoretically the ideal surgical procedure in every case of painful arthritis is the reshaping of the articular surfaces into their original contour, and the formation of a new joint, either with or without the interposition of some tissue such as fascia or fat, whereby the movements of the joint may approximate to normal. Many procedures have been described and much ingenuity has been shown in devising the correct technique required to obtain this ideal result, but unfortunately the average result of arthroplasty of the hip, however performed, has been decidedly poor.

In the series under discussion arthroplasty has been performed on 7 occasions during the past twelve years. In 5 cases the result was so indifferent that the patient eventually insisted upon some other treatment being adopted in order to improve the condition. In each case disappointment was expressed on account of the small range of movement possible in the joint, and also because even this range was not accomplished without discomfort. In 5 cases arthrodesis of the joint was necessary and was agreed to readily. The 2 remaining patients were satisfied, they were able to carry on their duties with comparative comfort, and although the result was not perfect, yet in each case the patient considered he was better than before the operation. The only movements possible were flexion and extension through a range of 30° and 40° , as the leg passed from the straight into the flexed position it also went into slight abduction and external rotation, but this was the only abduction or rotation possible to the joint. The X-ray photographs (*Figs 499, 500*), which were taken five years and four years after the arthroplasty, show the great heaping up of new bone round the joint which is the cause of this loss of rotation. None of these cases of arthroplasty can be classified as anything but a failure.

4 Pseudarthrosis—The operation of pseudarthrosis, or the formation of a false joint at some distance from the affected joint, is, however, in an entirely different category. It is an operation designed to give movement where movement is essential, as in a case of spondylitis in which one or both hip-joints are also affected, or when both hip-joints are involved and useful movement is not possible. It has been carried out on 4 occasions in this series, and in each case movement has been obtained, although usually at the expense of a certain amount of stability. This want of stability is the one great drawback to the wider use of this procedure. The operation is performed by removing a large portion of the neck and upper end of the shaft of the bone, and then implanting the great trochanter—to which the muscle group is still attached—on to the head and remaining portion of the neck. The upper divided end of the shaft of the femur now lies below the intumed outer surface of the great trochanter, which is completely covered by muscle attachments.

and therefore cannot re-unite with the shaft of the bone. Such a procedure invariably gives movement, but owing to the loss of control of the gluteal muscles on the shaft of the bone there is an inevitable diminution in the voluntary power of the limb.

There are two definite indications for the operation: firstly, bilateral osteo-arthritis in which the legs have become so adducted that they cannot be separated, or may even actually cross each other, and secondly, when the lumbar spine also is involved in an extensive osteo-arthritis. When both joints are involved it will often be found that one hip is painless and firm while the other shows a small range of painful passive movement. If the operation is carried out in such a case, it should be performed on the painful side, leaving the painless limb as a sound



FIG 499 —The result of an arthroplasty performed five years previously, showing the heaping up of bone on the rim of the acetabulum and consequent limitation of movements



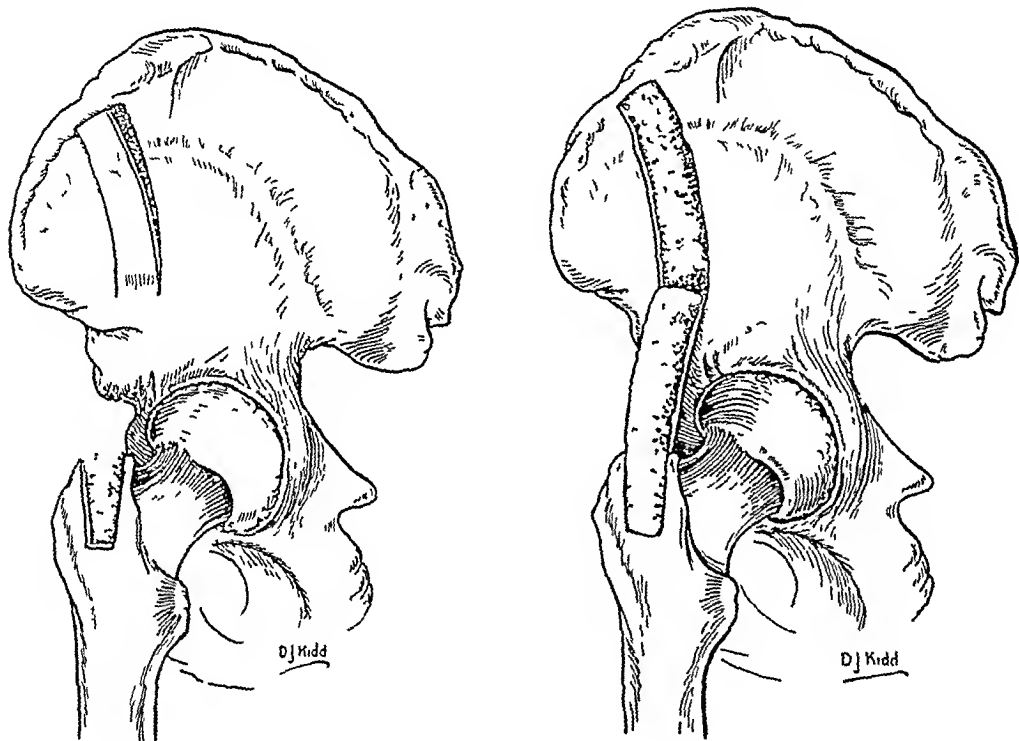
FIG 500 —Similar result of an arthroplasty after four years

weight-bearing pylon. Again, in some cases it is found that with bilateral affection one leg is definitely longer than the other, and here the indication is clear and the operation should be carried out on the longer side.

5 Arthrodesis —When arthroplasty failed in the cases reported in this series we fell back instinctively on the operation of arthrodesis, and in all cases this gave a good result without pain, and with full stability. It might seem that this operation is the best for every case of painful monarticular osteo-arthritis of the hip-joint because it eliminates all pain from the joint, retains full stability of the limb, and leads to no shortening of the leg.

Including the 5 cases where an arthrodesis was performed following the failure of an arthroplasty it was performed on 17 cases in this series, and the patients were

highly satisfied with the result. It was not successful from the point of view of producing a bony ankylosis in 6 cases. These were all included in the group of 10 in whom an intra-articular arthrodesis alone was employed, so that of these 10, a bony union was secured in only 40 per cent, but in each of these 6 cases, although there was 5° to 10° of passive movement, the clinical result was excellent. Bony union was secured in each of the other 7 cases by means of the combined intra- and extra-articular arthrodesis, where, in addition to a complete removal of all the cartilage from the head of the femur and acetabulum and a wide-spread fragmentation of the cancellous bone underneath, a flap of ilium above the level of the hip-joint was turned down and attached to the femur in the region of



FIGS 501, 502—Extra- and intra articular arthrodesis

the great trochanter (Figs 501, 502). Its base still formed part of the structure of the wall of the ilium and its blood-supply was preserved by this living continuity.

This method of combined operation makes for a more certain result (Fig 503), and it is preferable to the single method where the head of the femur—which has been diminished in size—is placed in the much enlarged acetabulum, thereby giving a small area of bone-to-bone contact.

Although the operation of arthrodesis gives complete relief to the pain and deformity of the hip and the patient is highly pleased, it is not without its disadvantages. The following case illustrates this very well. The patient was suffering from a very extensive painful osteo-arthritis of the right hip, and in January, 1926, an arthrodesis was performed. Six months later she was able to walk freely and without pain, and this state of relief lasted for six years, until 1932, when she returned complaining of pain and aching in the lower lumbar spine, undoubtedly

due to the increased work thrown on this area by the absence of movements in the hip-joint. Unfortunately this is not an isolated case, as in 6 others there was a similar complaint of lower lumbar and sacro-iliac pain which was not present before the operation. The operation also is one of some magnitude, as it requires thirty-five to forty minutes at least for its accomplishment, and the lifting out of the head and cleaning out the acetabulum involve considerable shock.

6 Bifurcation—The question of shock must be very seriously considered in those patients who are fat with weakened cardiac muscles, as such an extensive surgical procedure may be quite beyond their powers of resistance. A single case may be taken to illustrate the importance of this point. A lady 52 years of age



FIG 503—Result of an intra- and extra-articular arthrodesis of hip

and 13½ stones in weight, had been under the care of a physician for the previous four years on account of slight myocardial degeneration. She developed an extremely severe condition of osteo-arthritis of one hip so that she was unable to walk more than a few steps in her bedroom. As she was so exceedingly stout it was impossible to fit her with any form of hip shield or plaster spica, and it was decided that if at all possible some form of surgical treatment was advisable. After consultation it was decided that for her the only safe anaesthesia was gas and oxygen, which she might stand for not more than fifteen to twenty minutes. The operation of Lorenz bifurcation was successfully employed on this occasion, and she is now able to walk without pain for a quarter of a mile, and her general health is much improved as a result of the exercise which the operation has rendered possible. It is now largely owing to this consideration of absence of shock that I perform the operation of Lorenz bifurcation in certain suitable cases of non-articular osteo-arthritis, and this procedure has been carried out on 15 occasions.

The operation has many great advantages, it can be performed in a few minutes, the shock is negligible, and the recumbency after the operation need not be so long or so continuous as it must be after arthrodesis of the joint

Technique—An incision 6 to 7 in in length is made from the tip of the great trochanter along the line of the femur, and the muscles in this area are partially stripped by blunt dissection so that the level of the neck and head of the femur may be ascertained. An oblique osteotomy is now made in the shaft of the femur, passing from below upwards and inwards at such a level that the inner end of the lower fragment lies about $\frac{1}{2}$ in below the level of the head of the femur (Fig 504). The upper end of the lower fragment is now displaced inwards and slightly upwards by means of a smooth lever and by abduction of the leg so that the shaft of the femur comes to

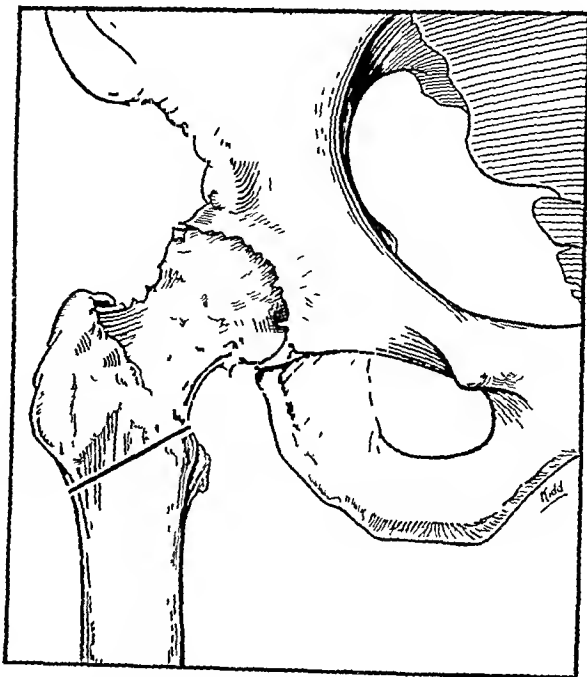


FIG 504—Diagram showing site of osteotomy for Lorenz operation

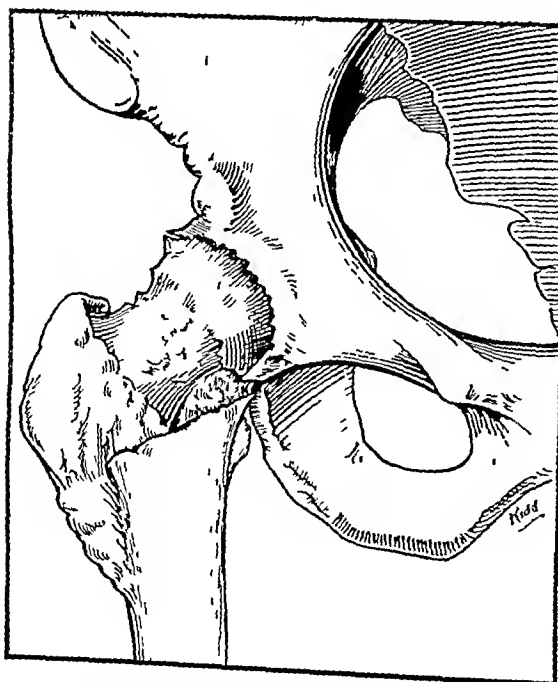


FIG 505—Diagram showing position of the fragments after a successful Lorenz osteotomy

lie just below the lower border of the acetabulum (Fig 505), and unless this position of the shaft is obtained the result will be unsatisfactory. In this situation the shaft of the femur lies outside the capsule of the joint and there is no fear of union occurring between the shaft and the acetabulum. Originally this operation was performed as a subcutaneous osteotomy and the final position of the lower portion of the bone was left largely to chance. This is the most common source of failure, and the operation should always be carried out through an incision sufficiently long for a careful inspection of the whole site of operation, and for the verification of the correctness of the final site of the femoral shaft.

There are certain essential

conditions for success in this operation (1) The new site of the upper end of the shaft of the femur must be just below the edge of the acetabulum, (2) There must be union between the portions of the divided femur, as otherwise a weak painful hip-joint is the invariable outcome. After the division of the bone the lower fragment is pushed inwards into its new position, no effort is made to change the position of the upper fragment, which tends to follow after the lower fragment as the two fragments have so many muscular attachments in common. The leg is then fixed in plaster with the hip neither abducted nor adducted, and after a suitable rest of four to five months in plaster—during the latter two months of which time the patient is allowed to get about with weight-bearing—the plaster is removed and complete freedom is allowed. The two



FIG. 506—X-ray of a case of osteo arthritis of hip before a Lorenz osteotomy was performed



FIG. 507—X-ray of case shown in Fig. 506 two years after a Lorenz osteotomy was performed. The patient is entirely free from pain and has been at work in a shop for the last year

portions of the femur have now joined, movement which may have been present in the joint is not lost but is taking place through a different angle, and the relief is almost as great as after the operation of arthrodesis, without many of the very great disadvantages of this operation (Figs 506, 507)

Results—In 12 cases the result has been excellent, the pain has gone, movement although reduced is still present, and there is not the same tendency to strain the lumbar and sacro-iliac regions. In my opinion the operation holds a very important place in the treatment of osteo-arthritis of the hip-joint. In 3 cases the results have not been satisfactory, pain was not removed and deformity was still present (Fig 508). The reason in each case was the same—namely, the operation incision was too small, so that the whole area could not be inspected and the

shaft was not placed in the correct position. As a result body weight continued to pass through the hip-joint as before. This error can be prevented by a larger incision, by inspecting the area of operation, and making sure of the final situation of the upper end of the lower fragment of the femur. If this is done, the result will almost certainly be satisfactory.



FIG 508—X-ray of an unsuccessful result following a Lorenz osteotomy, showing the shaft not displaced far enough under the acetabulum and failure of correction of the line of weight-bearing.

CONCLUSIONS

Manipulation may produce a relief of symptoms and increased movement of a temporary nature in cases of osteo-arthritis.

Arthroplasty is the ideal operation, but at present our methods and technique fail to secure any real benefit for the patient, and the operation is usually a failure of the joint, but it is a very extensive procedure, and, if successful, results in a great increase of strain on the lumbar spine and sacro-iliac region.

Arthodesis removes the pain and deformity and greatly increases the stability of the joint, but it is a very extensive procedure, and, if successful, results in a great increase of strain on the lumbar spine and sacro-iliac region.

The bifurcation operation of Lorenz is simple, of short duration, and if performed correctly leads to a relief of pain and deformity with no loss of stability, and does not cause strain on the lumbar region.

THE PATHOGENESIS OF PAGET'S DISEASE OF THE NIPPLE AND ASSOCIATED LESIONS

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DURING the last few years I have been engaged in a study of Paget's disease and other affections of the breast and have published several papers (1927-34) dealing especially with the former condition. It may, however, be well to give a short general account of my views and the observations on which they have been based, especially in relation to the surgical aspect, and in doing this I have added some new observations along with some new illustrations. I have not made an attempt to deal with the extensive literature on the subject.

In my first paper (1927) reasons were given for coming to the conclusion that in the great majority of cases Paget's disease results from an extension of cancerous proliferation from the ducts of the nipple ('intra-duct carcinoma') to the surface epidermis of the latter—an overflow of cancer cells, as it were, to the epidermis with subsequent spread and multiplication therein. I have said "in the great majority of cases" because I admitted the possibility of the occasional occurrence of a primary epidermal carcinoma with intra-epidermal spread such as is met with on other parts of the body. This, however, is referred to below.

To the surgeon the most important practical point is the tendency for Paget's disease to be associated with or followed by carcinoma in the substance of the breast, and the relation of these two occurrences has often been the subject of discussion. In connection with this it should be observed that in his original paper Paget not only gave an accurate account of the naked-eye appearances of the nipple lesion but noted that carcinoma frequently ensued and that the site of the malignant growth had no relation to the nipple, but might be deep in the substance of the gland. (As a matter of fact, carcinoma developed in all his cases.) What, then, is the relation between the Paget lesion and the carcinoma? If the former is a primary lesion of the epidermis, I can see no possibility of any causal relationship. If, on the other hand, they are both secondary to the same condition—namely, intra-duct carcinoma—their association receives at once a ready and an intelligible explanation. Accordingly, if we use the term 'Paget's disease' in a sense more in conformity with his original account—a characteristic nipple lesion *liable to be associated with mammary carcinoma*—I believe that the association of the two conditions, Paget's disease and carcinoma of the breast, is due to both being sequels to antecedent malignant proliferation within the ducts of the gland. When the Paget lesion is present the surgeon should have in his mind the picture of this proliferation within the ducts—a proliferation which may break through the walls at any time and give rise to an ordinary infiltrating carcinoma, if in fact this has not already occurred.

As the Paget cells in the epidermis are the characteristic feature of the disease I may anticipate what follows by stating at this point that similar cells are met with within the epithelium both of the ducts and of the acini. In all these situations their appearance and behaviour are essentially of the same nature. The only important difference is that the Paget cells in the epidermis undergo degenerative changes as they pass or are carried towards the surface and thus become the well-known small and sharply contoured structures. In the epithelium of the ducts and acini, on the other hand, they remain in a more healthy and active state and resemble the active Paget cells in the deepest layers of the epidermis. We may thus give a general definition of a Paget cell as *a cancer cell growing within a healthy or at least non-neoplastic epithelium*.

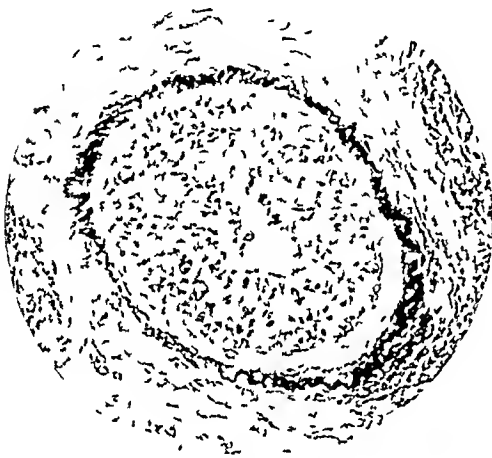
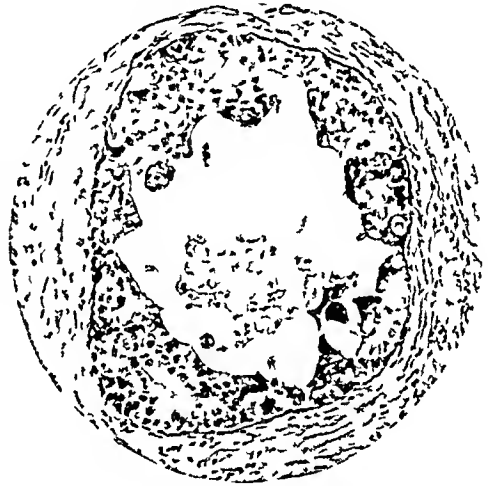
As in my view intra-duct carcinoma is a condition antecedent both to Paget's disease and ordinary carcinoma, it will be convenient to consider first its main features—its characters, modes of spread, and secondary results.

OCCURRENCE AND CHARACTERS OF INTRA-DUCT CARCINOMA

I have applied the term 'intra-duct carcinoma' as meaning a malignant proliferation of duct epithelium before it has broken through the normal limits, i.e., the walls of the ducts, and infiltrated the tissue spaces. The term seems to me a justifiable one and the most convenient. The use of the term 'carcinoma' may appear misleading, but it should not be so if the qualifying adjective is kept in view. As I have said elsewhere, epithelial cells do not become malignant on getting into the tissue spaces, they invade the latter because they have acquired the essential characters of malignant neoplasia, and they acquire this character before they transgress the normal boundaries. It is true that the sure sign of malignancy is infiltration, but when one keeps in view the two facts—namely, that the malignant property is developed before actual infiltration occurs, and, secondly, that the histological features of the cells in question correspond whether they are within the ducts or outside in the tissue spaces—then the term 'intra-duct carcinoma' is both justifiable and of descriptive significance. It is preferable to the term 'duct carcinoma', as the latter may signify merely an ordinary carcinoma which has arisen from the epithelium of the ducts.

Intra-duct carcinoma may be regarded as the final stage of epithelial hyperplasia often met with in chronic breast disease and often associated with a varying degree of fibrosis and cystic change. The stages of such epithelial hyperplasia have been described in detail by Cheate (1925-31) and by Charteris (1930), and I agree in all essential points with their accounts. It is sufficient to say that all transitions from simple papillomatous ingrowths in the ducts to intra-duct carcinoma are found. We meet with papillomata with broad connective-tissue base and also those with filamentous supporting connective tissue, but in both types there is always co-ordinated growth of epithelium and connective tissue and the normal relationship of these is maintained. Further stages are those in which the stroma gradually diminishes in amount until one meets with epithelial proliferation without any connective tissue between the cells. The cells may form definite projections from the wall of the duct, or again they may form masses in which polarity of the

cells is still shown by the formation of acinus-like circles, or, lastly, the cells may have no definite arrangement. The cells are at first fairly uniform in size and in their nuclear characters, but later show great variations both in size and in form. The nuclei also come to vary, many are enlarged, pale, and of vesicular type, whilst aberrant forms also are met with. Ultimately the duct may be filled with a mass of cells showing all the histological characters of encephaloid carcinoma (Figs 509, 510). It is to this stage of epithelial hyperplasia that I consider the term 'intra-duct carcinoma' may properly be applied. It is quite evident that such cells may break through the walls of the containing duct and infiltrate the tissues, thus giving rise to ordinary carcinoma of the breast, and, as a matter of fact, the actual break through may occasionally be seen.

FIG 509 ($\times 75$)FIG 510 ($\times 90$)

FIGS 509, 510—Transverse section of two intramammary ducts, the seat of intra duct carcinoma, showing characteristic appearances. In Fig 509 there is some hyperplasia of the elastica.

It may also be mentioned that the cells undergoing malignant proliferation may extend from the small ducts into the acini and may grow within the latter. In this way there may occur what is practically a replacement of the epithelium of the acini by cancerous growth, the normal structural outlines of the acini being still maintained. We may then speak of an *intra-acinous carcinoma*. I have seen such a condition on an extensive scale without there being any actual infiltration of the connective tissues.

I may refer to one point in connection with the interpretation of the changes described. In cases where ordinary carcinoma is present and the ducts also are filled with cancer cells, the view has been taken by some writers that the ducts have been invaded by cancer cells. The possibility of such an occurrence as an occasional event cannot, of course, be denied, but I have no doubt that it is exceptional, and that the sequence of events is of the opposite kind—the cancer outside is secondary to the disease within the ducts. The following reasons for this view may be given. In a case where ordinary carcinoma is present the various stages of evolution of intra-duct carcinoma may sometimes be followed, epithelial hyperplasia terminating

in malignancy may be seen. We can scarcely suppose that cancer cells after penetrating ducts from outside lose their anaplastic characters and become more differentiated again. But more conclusive evidence is that intra-duct carcinoma may be present, and often is, without any infiltrating carcinoma of the tissues being discoverable. I have seen the combination of extensive intra-duct carcinoma in the breast with spread to the acini, intra-duct carcinoma of the nipple and Paget's disease, without any infiltration of the connective tissue. Or again, and this is a stage more frequently met with, ordinary carcinoma may be associated with intra-duct carcinoma in one area while intra-duct carcinoma alone is present in other parts of the breast, and, further, may show different stages of evolution. I consider, therefore, that in the vast majority of cases where intra-duct carcinoma and ordinary carcinoma are present, the latter is secondary to the former.

There is no doubt that intra-duct carcinoma is a condition of frequent occurrence. I have found it in the large majority of cases of ordinary breast carcinoma which I have recently examined, and, in view of the fact that a small area showing the lesion may escape detection, I have formed the opinion that it is usually present. Its distribution varies greatly. It may occur in a localized area, often attended with fibrous induration and cyst formation, or several such areas may be present, or again it may affect a considerable part of the breast. When it is present in some ducts, epithelial hyperplasia at various stages of evolution may be present in others. It may affect the ducts of the nipple and those in the underlying tissue as well as in the breast itself, and occasionally the former may be affected alone. It will be clear from what I have stated that it is only when the upper parts of the ducts or of a duct in the nipple are affected that Paget's disease occurs as a secondary phenomenon. It may be definitely stated also that intra-duct carcinoma, the final stage of epithelial hyperplasia, occurs at an earlier period of life than does ordinary carcinoma. This has been insisted on by Cheate, and I agree fully with him. As an illustration, in the five cases of Paget's disease on which I based my original views and in all of which duct carcinoma was present, the age of the eldest patient was 48 years.

It is clear, too, that intra-duct carcinoma is a very chronic condition and may exist for many years without breaking through the walls of the ducts, it may, in fact, never break through at all. Further, in a recent paper (1934) I have shown that in some cases the growth may at places undergo retrogression and disappear, this process being accompanied by overgrowth of the connective tissue internal to the elastica of the duct wall, complete obliteration sometimes following. If my views with regard to Paget's disease are correct, then in cases where this disease is present we get another indication of the chronicity of the intra-duct carcinoma which precedes it for a considerable time. For example, I have recently met with a case in which the Paget lesion was present for ten years. No ordinary carcinoma could be found after careful microscopic examination, but extensive intra-duct carcinoma was present, and in some of the ducts affected by it obliterative changes had occurred.

To sum up, then, intra-duct carcinoma is a common affection of chronic nature, its time of incidence preceding the cancer age by a considerable period. It often breaks through the normal confines of the ducts and gives rise to ordinary carcinoma, but may exist for a long period without doing so, occasionally it leads to the relatively uncommon condition of Paget's disease.

MODES OF SPREAD OF INTRA-DUCT CARCINOMA

This is a matter of much importance in relation to Paget's disease. I have already stated that malignant proliferation within ducts varies much in its distribution. It may occur in different parts of the breast, may affect considerable areas, may occur in the ducts of the nipple or of the breast or of both together. We have thus to consider how this varied distribution is brought about. There appear to me to be three main factors concerned —

1 In the first place, the malignant change may arise in multiple foci, of this there can be no doubt. Thus it is met with in different parts of the breast which are anatomically distinct and is also met with in several ducts in the nipple and beneath it, occasionally in a considerable proportion of these ducts. We know nothing of the essential cause or influence which acts on the epithelium and gives rise to the malignant change, but, whatever be its nature, it may clearly act on different portions of the breast at the same time.

2 In the second place, when the epithelial cells have assumed the neoplastic character they may grow in masses within the ducts and come to form a sort of injection of them, occasionally the same is met with in the acini.

3 But, in the third place, there is another mode of spread, namely, a spread of cells with malignant properties in relatively healthy epithelium—the *intra-epithelial spread of carcinoma*. This is an occurrence which is not in any way peculiar to the disease under consideration, but is seen in other conditions where the true nature of the phenomenon does not admit of any doubt. This process of intra-epithelial growth is in my opinion all-important in connection with the changes which we are considering, and some facts with regard to it may now be given.

By intra-epithelial growth, then, we mean the invasion of normal epithelium by cancer cells, the latter grow within the epithelium and destroy it to a varying extent, the epithelium playing a passive rôle. The cancer cells may occur in masses in the invaded epithelium or they may be disseminated and occur singly, this latter condition being apparently due to their wandering in the epithelium. Such a mode of growth is now coming to be generally recognized. I have elsewhere written on the subject (1930), but the main facts with regard to its occurrence may be summarized as follows —

1 In the first place, carcinoma may start in an epithelium and grow within it. This is best illustrated by primary intra-epidermal carcinoma about which much has been written by dermatologists in recent years. Bowen's disease is one well-recognized example, but there are other varieties. The cells of the growth are distinguishable from the normal epithelium within which they grow in masses or in a scattered form, they present different morphological features and there may be aberrations in type. The growth is of a very chronic character, often going on for years, occasionally infiltration of the cutis vera follows, an epithelioma, basal-cell carcinoma, or a growth of other type resulting. Primary intra-epidermal carcinoma, though uncommon, can no longer be regarded as a rarity.

2 Secondly, a malignant growth infiltrating the cutis may invade the epidermis and spread within it, this is a rare occurrence, but when it is met with the appearances are of striking nature. Carcinoma of the breast may occasionally invade the epidermis in this way, and the invading cells then behave in a fashion analogous

to what is seen in Paget's disease. The most striking example I have seen of such a secondary epidermal invasion is that described by Shaw Dunn (1930) in the case of a mucoid (colloid) carcinoma of the rectum. In the drawing accompanying his paper the cancer cells, stained red with mucicarmine, are clearly picked out from the epidermal cells and their distribution is seen at a glance. No clearer or more convincing demonstration of what I must call the wandering of the cancer cells could be had. A similar phenomenon is occasionally seen in malignant melanoma, as has been described by others. I have recently seen such a case, the growth being at the anal orifice, and the pigment-forming malignant cells are distributed in the epidermis just as in Shaw Dunn's case.

3 In the third place, the epidermis of the nipple may be invaded by cells of an intra-duct carcinoma in the upper part of a duct of the nipple—the route of spread in this case is an intra-epithelial one throughout. This I hold to be the ordinary mode of origin of Paget's disease, and it may now be considered more in detail.

INTRA-EPITHELIAL SPREAD OF CARCINOMA OF THE BREAST AND ITS RESULTS

In giving an account of this subject, it seems a good plan to start with the changes in the ducts in the breast and trace them to the surface of the nipple. It has already been stated that intra-duct carcinoma is a common primary event in malignant disease and that in its spread intra-epithelial growth may be concerned.

This is shown by the presence of cancer cells growing in healthy epithelium where intra-duct carcinoma is spreading. The cells are often large and of rounded or oval form with vesicular nuclei and they occur in small collections and singly. In many cases they are sharply marked off from the invaded cells which become pressed upon and atrophied, one can often see the nuclei of the latter flattened and degenerating between the cancer cells. At a later stage of growth a duct may be lined by a sheet of cancer cells several layers in depth, whilst the fact that the growth is really intra-epithelial is shown by a surviving layer of the original epithelium on the luminal surface. Intra-epithelial growth may also be found in the acini of the breast, and here the large pale cancer cells can be clearly seen within the otherwise unchanged epithelium (*Fig 511*).

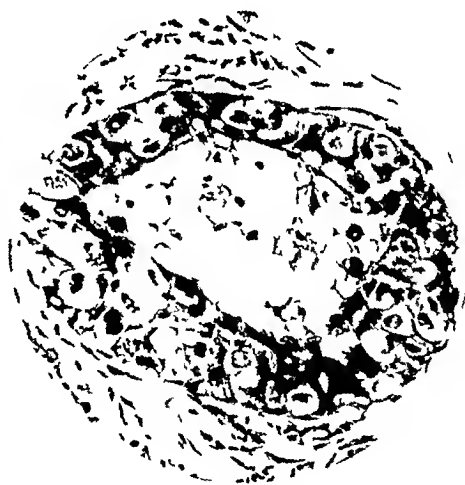


FIG 511—Mammary acinus, the seat of intra epithelial growth of cancer cells. The cancer cells ('Paget cells') with clear protoplasm are seen growing in the epithelium of the acinus ($\times 200$)

Occasionally, collections of acini are replaced by groups of cancer cells still within normal confines, they are usually surrounded by an abundant collection of lymphocytes and plasma cells. A study, then, of the breast alone, quite apart from Paget's disease, shows that the epithelium, both of the ducts and of the acini, may be invaded by cancer cells which have not

yet grown out into the tissue spaces. Such a mode of growth corresponds with that in the epidermis of the nipple now to be described and is in conformity with the definition of a Paget cell given above. Other illustrations of intra-epithelial growths in ducts and acini will be found in my first paper and in a paper by Simard (1930).

Spread to the Epidermis—If intra-duct carcinoma spreads thus within the epithelium of ducts and acini, an important question comes to be: What will happen when the growth reaches the orifice of a duct? Let us keep before us a picture of the histological features at this position. The columnar epithelium at the orifice of a duct is, of course, continuous with the deeper cells of the epidermis and there is a transition between the two, sometimes relatively abrupt, sometimes more gradual. The transition, too, occurs at varying levels, the stratified squamous

epithelium sometimes extending for some distance down the duct. When the growing cancer cells reach this junction, as often occurs, there are three possibilities: (1) The growth of cancer cells comes to an end, (2) The cancer cells may break through into the cutis and give rise to an infiltrating carcinoma, (3) The intra-epithelial growth may continue. With regard to the first possibility, one can say nothing. One may sometimes trace an intra-duct carcinoma only up to the orifice of the duct, but one cannot say that the growth has stopped. Again, the possibility of ordinary carcinoma arising at this site cannot be denied, but I have never seen any trace of this. The third possibility is the ordinary occurrence.

The intra-epithelial growth of cancer

cells goes on, and of necessity the cancer cells pass from the duct epithelium into the epidermis. Thereafter they spread and produce all the features of Paget's disease.

The relation of the cancer cells invading the epidermis to the resulting effects needs only a short account. The cells are in a resisting tissue, a relatively unfavourable environment, and they spread slowly. They can flourish only in the more cellular layers and especially in the deep part of the stratum Malpighi (*Fig 512*). As the cells of the epidermis grow and move towards the surface the Paget cells are carried with them, the latter degenerate as they move upwards and come to form the sharply contoured structures so well known. The Paget cells spread widely in the epidermis over the nipple and grow both in clumps and as widely scattered solitary cells. The latter appearances, as I have indicated above, can be explained only on the view that they actively wander. Sometimes the cells are large and show active division, mitoses being present. They often correspond closely in appearance with the cancer cells in the upper extremities of the ducts. Sometimes,



FIG 512.—High-power view of deep portion of epidermis of nipple in Paget's disease. Note the large Paget cells of active appearance invading the rete Malpighii and sharply defined from the cells of the latter ($\times 600$).

however, nearly all the Paget cells are in a more or less degenerated condition, and then from such a case alone it would not be possible to interpret the appearances. It is only by studying a series of cases that one can trace the various modifications which the cells undergo.

As a rule the Paget cells are oval or rounded, in fact quite anaplastic, but occasionally one sees a return to a columnar type. It is not uncommon to find traces of this in the deeper layers of the epidermis, where the invading cells may form an imperfect layer resting on the cutis. Evidence of this is shown in *Fig 513*, but *Fig 514* is a more striking example. The columnar epithelium in the latter encloses a small space containing fluid, the epidermal cells being raised above it. We have really an example of a small metastasis of adenocarcinoma, the cells of

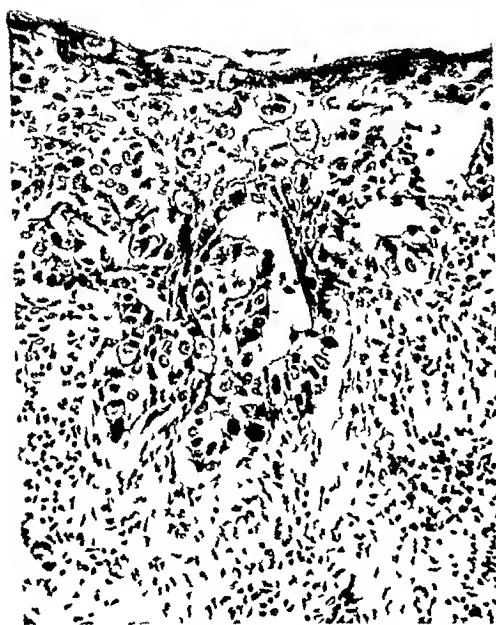


FIG 513—Paget's disease of nipple. In the deeper part of the epidermis the Paget cells show transitions to the columnar type ($\times 200$)



FIG 514—Paget's disease of nipple. The Paget cells form a distinct layer of columnar type resting on the connective tissue, with a space containing fluid above them ($\times 200$)

which have reached the epidermis by the intra-epithelial route. In other parts of the epidermis all transitions to Paget cells of ordinary type were present. Such appearances seem to show clearly that the malignant cells are not of epidermal origin and confirm the view which I have put forward.

As soon as the malignant cells are present in any part of the epidermis a cellular reaction occurs in the connective tissue below. There is marked accumulation of lymphocytes and plasma cells, we find also new formation of blood-vessels and increased vascularity. The invading cells in some way irritate the tissue and a certain amount of exudation on to the surface of the epidermis occurs. These changes result in the red and raw-looking surface and the serous discharge characteristic of the disease.

Since my first paper was published I have examined about twenty cases of Paget's disease of the nipple, and observations on these have merely confirmed the view originally expressed. In all of them intra-duct carcinoma was present in the nipple, and in a large proportion direct spread of the carcinoma from the ducts to the epidermis could be traced. As a striking illustration of this, reference may be made to what was found in one case. In *Fig 515* there is shown a duct of the nipple cut longitudinally in its whole length with the exception of a small portion at its upper end, the depression in the epidermis indicates the position of the orifice. The duct throughout the portion shown is filled with cancer cells, and in the deepest part of the epidermis at the depression there are several small collections of large pale cells. A high-power view of these cells is shown in *Fig 516*. As is clearly seen, they are large and appear pale and present a sharp contrast



FIG 515—Longitudinal section of two ducts in nipple. The one on the left is the seat of intra-duct carcinoma and is almost filled with cancer cells. A few cancer cells in small groups—Paget cells—are present in deep part of epidermis at duct orifice (cf *Fig 516*) ($\times 10$)



FIG 516—High power view of deep portion of epidermis at orifice of duct shown in *Fig 515*. Note the collections of Paget cells with clear protoplasm sharply marked off from the epidermal cells. The Paget cells were present only in this situation ($\times 200$)

to the cells of the epidermis. They were found to correspond in all histological details with the cancer cells in the duct. We have here a picture of the earliest implication of the epidermis, in fact the cancer cells have just arrived in its deepest part. It does not appear to me possible to interpret the appearances shown in these figures in any other way. It may be added that there had been in this case no suspicion of the presence of a Paget lesion, and further that no Paget cells could be found at any part of the epidermis except at the orifice of the duct.

As stated at the outset, I do not deny the possibility of the occurrence of primary intra-epidermal carcinoma of the nipple such as is sometimes met with on other parts of the skin surface. I have met with only one case of this kind and

it was in a male subject The underlying glandular tissue showed no lesion, and the significant fact is that a rodent ulcer had developed in connection with the skin lesion, an occurrence which is recognized in connection with such growths elsewhere On the other hand, I have never seen basal-cell carcinoma or epithelioma or any form of invasive epidermal growth arising in connection with Paget's disease of the female breast

SUMMARY

From the above account it will be seen that in the evolution of Paget's disease there are two main factors concerned, namely, *intra-duct carcinoma* and the *intra-epithelial mode of spread of this growth* The main features of intra-duct carcinoma, a lesion of frequent occurrence, and its varying distribution have been described The intra-epithelial mode of spread occurs in the epithelium of the ducts and acini as well as in the epidermis of the nipple—the 'Paget cells' are cancer cells growing in non-neoplastic epithelium Paget's disease occurs only when the intra-duct carcinoma is present in the upper portions of the ducts in the nipple, and is due to the spread of the cancer cells from ducts to the epidermis by the intra-epithelial route Intra-duct carcinoma in this situation is relatively uncommon, and hence Paget's disease is somewhat rare Much more frequent is the occurrence of intra-duct carcinoma in the ducts within the mamma, and then the ordinary result is a direct break-through of the cells with the production of an ordinary infiltrating carcinoma Intra-duct carcinoma may lead to one or both lesions, and thus Paget's disease may precede or may follow ordinary mammary carcinoma, or again may develop without the occurrence of the latter

BIBLIOGRAPHY

- CHATERIS, A A, *Jour Pathol and Bacteriol*, 1930, xxxiii, 101
 CHEATLE, G LENTHAL, *Brit Jour Surg*, 1925-6, xiii, 509
 CHEATLE, G LENTHAL, and CUTLER, M, *Tumours of the Breast*, 1931 London
 DUNN, J SHAW, *Jour Pathol and Bacteriol*, 1930, xxxiii, 1297
 MUIR, R, *Ibid*, 1927, xxx, 451
 MUIR, R, *Ibid*, 1931, xxxiv, 594
 MUIR, R, *Brit Med Jour*, 1930, ii, 587
 MUIR, R and AITKENHEAD, ANNE C, *Jour Pathol and Bacteriol*, 1934, xxxviii, 117
 SIMARD, CH, *Bull Assoc franç pour l'Etude du Cancer*, 1930, xix, 50

PARAPLEGIA IN POTT'S DISEASE, WITH SPECIAL REFERENCE TO THE PATHOLOGY AND ETIOLOGY*

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PARAPLEGIA is the commonest and most distressing of the graver complications of tuberculous disease of the spine. To the patient the onset of 'paraplegia' seems to herald his worst forebodings. Percivall Pott,²⁶ writing in 1779, summed up the situation from the layman's point of view in a single sentence which needs no amplification to-day: "If the patient be an infant, it becomes an object of constant though unavailing distress to its parents, if an adult he is rendered helpless to himself, and useless to all others, which of all possible states is surely the worst."

SOURCES OF CLINICAL MATERIAL AND STATISTICAL SURVEY

Gross Incidence of Paraplegia in Pott's Disease—The gross incidence of paraplegia has been estimated to be as high as 20 per cent by some and as low as 5 per cent by others. Such observations will vary with the ages and the stage of the disease of the patients forming the bulk of any given series, and with the conditions under which they are seen, whether for a short time only or over long periods. For statistical purposes a survey has been made by the writer of 800 patients with Pott's disease from four different hospitals, forming at each a continuous series:

St Thomas's Hospital (in-patients)	(1922-32)	182
Pyrford Orthopædic Hospital	(1922-32)	82
Queen Mary's Hospital, Carshalton	(1927-32)	206
Shropshire Orthopædic Hospital, Oswestry	(1927-32)	331
Total number with Pott's disease		801

Among these 801 were 92 with paraplegia, giving a gross incidence of 11.4 per cent. For further analysis there have been added to this series of 92 patients a further 94 collected by H. J. Seddon from other hospitals, making a combined series of 186 patients with paraplegia. Seddon's 94 patients were collected from the Royal National Orthopædic Hospital and the country branch of this hospital at Stanmore, St Luke's Hospital, Lowestoft, Princess Mary's Hospital and the Royal Sea Bathing Hospital, Margate, and St Vincent's Orthopædic Hospital, Pinner. Among these 186 patients were 7 that had a second paralysis at some

*The theses from which this and the following article are taken were both awarded the Robert Jones Medal and Prize last year by the British Orthopædic Association. The authors have collaborated for the purpose of the present publication, having eliminated the duplication of those parts which were common to both theses.

time after recovery from the first, so that the combined series contains 193 paraplegias in 186 patients. Patients with paraplegia were also studied at the Royal Cripples Hospital and the Woodlands Hospital, Birmingham, and Addenbrooke's Hospital, Cambridge, but these fell into no continuous series. Material for pathological study was obtained from many of the above-mentioned hospitals and from the museums of pathology of the London Medical Schools.

Incidence of Paraplegia in Relation to Age—Tuberculosis of the spine has its onset much more commonly in childhood than in adult life. Onset is more frequent in the third than in any other year, and after the sixth year the incidence declines rapidly. According to Sorrel³¹ 64 per cent of all Pott's disease starts in childhood. As the onset of disease is commoner in childhood than in later life, it might be expected that paraplegia would also be more common in children. The paraplegia may, however, supervene many years after the onset of disease, and actually it occurs in this series of 188 patients about equally under and over the age of 16.

Incidence of Paraplegia in Relation to the Level of the Disease in the Spine—Paraplegia is rare with disease below the level of the 1st lumbar vertebra, since the cord has terminated below this point. Thoracic disease is far more common than cervical, and therefore the majority of paraplegias accompany thoracic lesions. In this series of 186 patients with paraplegia the level of the lesion was as follows—

Cervical (including disease the lower part of which involved the cervico-thoracic junction)			16
Thoracic	High	22	157
	Mid	83	
	Low	52	
Thoraco-lumbar junction			12
Lumbar			1
			<hr/> 186

Paraplegia with thoracic disease therefore makes up 84.4 per cent of all paraplegia, in this series.

Most writers state that paraplegia is not only commonest in thoracic disease, which is itself far commoner than disease at other levels, but also relatively commoner with disease in the thoracic than at any other level. In the writer's personal series of 92 paraplegias it happened, however, to be relatively commoner with cervical disease (11 paraplegias in 40 patients, i.e., 27.5 per cent) than with thoracic (78 paraplegias in 376 patients, i.e., 20.7 per cent).

DIFFERENTIATION OF POTT'S PARAPLEGIA INTO CLINICAL TYPES

Paraplegia in Pott's disease presents much variability in clinical appearance. It varies in onset. It may be the first sign of the disease either to the patient or his friends. Another patient may have had disease of the spine as a child, leaving him with a kyphus but otherwise strong and well for forty years—and then a paralysis supervenes. The paraplegia varies in degree sometimes it is complete below a certain level, sometimes only very partial, sometimes asymmetrical on the two sides. The persistence of the paralysis may be for only a few months, or it may be permanent—without any tendency to recover under treatment.

No single type of paraplegia is characteristic of Pott's disease This variability accounts for the divergence of opinion that has existed in the past as to the etiology of the paraplegia, since dogmatic theories of causation advanced by one authority based on an experience of comparatively few cases may be quite unacceptable to another who has seen patients mainly of a different clinical type

By comparison of a large number of patients with paraplegia it is found, however, that they can be classified as belonging to one or other of certain distinct clinical types In recent years some rationalization of our ideas upon paraplegia has been attained in this way by Professor E Sorrel and Mme Sorrel-Dejerine³¹ working at the Hôpital Maritime at Berck-sur-Mer These writers consider that the majority of paraplegias in Pott's disease fall into one or other of two main types Their first type (A) is paraplegia with onset early in the disease As far as can be assessed the paralysis supervenes between the sixth and eighteenth month The progress of the paralysis is rapid and when fully established it tends to be complete Their second type (B) is paraplegia of late onset, that is to say, it develops at least two years—often many years—after the onset of the disease It develops but slowly and is seldom complete They mention also a third type (C), that of transitory paraplegia In essentials this is a subdivision of Type A, but a mild and ill-sustained paralysis These writers have sought to show, moreover, that there is a considerable difference in the pathological findings between Types A and B, and suggest that two different clinico-pathological types should therefore be recognized which, they claim, have an entirely different etiology

Clearly it is of great value to obtain a division of patients by clinical means into distinct types, if such types can be shown to have consistent differences in their etiology, since the only sound basis of treatment is an understanding of the causative factors in each individual case

The Sorrels' differentiation of "early onset" and "late onset" paraplegia has provided a basis for further investigation and it has been possible to produce a somewhat amplified, but nevertheless simple, classification of patients with paraplegia which seems justified on both clinical and pathological grounds, and which is therefore of value with a view to prognosis and treatment This classification is as follows —

Early Onset Paraplegia —

Type I—Paraplegia with early active disease arising, persisting, and diminishing in direct relationship to the activity of the tuberculous infection of the spine Typically early in onset, usually within the first two years of the disease Typically a complete paraplegia Typically recovers completely, if the patient survives

Type II—Paraplegia associated in onset with early active disease, but persisting permanently, even if the tuberculous infection in the spine has become completely quiescent In most cases indistinguishable in early stages from Type I Typically a complete paralysis at first, but frequently showing some degree of recovery before passing into a state of permanent paralysis

Late Onset Paraplegia —

Type III—Late onset paraplegia appearing at any time up to many years after apparent quiescence of the disease At the time of onset of the paralysis evidence of activity of the tuberculous lesion may or may not be present but is frequently lacking clinically Almost always only a very partial paraplegia

With the passage of time Type III paraplegia is found to fall into one or other of two subdivisions —

Type III a—Late onset paraplegia recovering under further conservative treatment as for active tuberculous disease of the spine, whether this is clinically present or not

Type III b—Late onset paraplegia not recovering under any form of treatment, whether active tuberculous disease of the spine is clinically present or not

If the 193 paraplegias in the 186 patients in the series are divided into these types the following figures are obtained —

<i>Early onset paraplegia</i> —	
Type I	76 (39.4 per cent)
Type II	21 (10.9 per cent)
<i>Late onset paraplegia</i> —	
Type III	96 (49.7 per cent)
(Type IIIa 58, Type IIIb 38)	

Reference to the mortality rate from paraplegia has been left until the clinical types have been defined, since the only type which has a large and closely associated mortality is Type I—paraplegia with early active disease. Late onset paraplegia rarely leads directly to a fatal issue. The mortality rate for Type I paraplegia in this series has been approximately 30 per cent.

TYPE I. PARAPLEGIA WITH EARLY ACTIVE DISEASE

THE CLINICAL PICTURE

Recognition of the Type—This consists of finding early active Pott's disease associated with the paraplegia. Generally Pott's disease has been recognized before the onset of paralysis. Sometimes the paralysis is the first trouble of which the patient complains. Even so, the presence of Pott's disease is generally shown by early angulation, local or referred pain, and X-rays.

Sometimes in adults, in whom bone destruction is slower than in children, disease may be present in the vertebral bodies for some time before there is bone collapse or external deformity, and especially is this so when the disease is confined at first to the posterior parts of the bodies. The best X-rays may then fail to show bone destruction, and it may be months before a lateral film reveals change in the posterior region of a vertebral body.

Disease arising in a lamina or one of the postero-lateral intervertebral joints may likewise give rise to paraplegia without apparent X-ray change. Both are rare, except in suboccipital disease. All such paraplegia without X-ray change is rare, and is indistinguishable from that due to any extramedullary tumour. This will be referred to again later as the 'spinal tumour' type of paraplegia with early active disease. (See also p. 776.)

Onset—The onset of Type I paraplegia is almost always within the first two years of the disease, usually at or just before the time the spinal disease reaches its fullest development.

Progress.—The progress of Type I paraplegia is usually rapid, being further accelerated by lack of treatment. Muscular weakness, inco-ordination, and spasticity usually appear first, and progress to a paraplegia in extension, with increasingly frequent attacks of general flexor spasm. As the paralysis deepens it may become a paraplegia in flexion. In the worst cases of all—especially if treatment is

neglected, though sometimes in spite of it—much spasticity may be lost and the paralysis may become almost completely flaccid. With such the outlook is always grave, should recovery commence, its first sign will be a return of spasticity. Although motor changes are often the first to appear they are sometimes preceded by changes in sensation. In distribution sensory changes are variable, generally the extremities are affected first and most completely. There is no constant rule as to the order of loss of the various types of sensation. Vibration sense and deep pressure sense are, however, usually the last to disappear. When the paralysis is fully developed there is typically a zone of hyperæsthesia at the upper border of the affected area. Disturbance of sphincter control is often a late feature. Occasionally it is the first sign. Retention of urine and fæces occurs at first. Later automatic action of the sphincters may become established, or, in the most severe cases, the sphincters are completely paralysed and exert no control.

With deepening paralysis much vascular and 'trophic' change may be present in the extremities and in parts exposed to pressure, e.g., the skin of the back, making the avoidance of pressure sores difficult.

A transitory paralysis, lasting for perhaps only a few weeks, and rapid both in onset and recovery, occurs occasionally. It is most frequently seen at an early stage, when the patient is still walking about, and recovery is then frequently simply a response to putting the patient at rest with fixation.

Some degree of disturbance of cord function, as shown by weakness or absence of the abdominal reflexes or by an increase in the tendon reflexes in the legs, occurs in about 50 per cent of children with cervical or thoracic caries at some stage in their illness, but cannot be classed as paraplegia. This is less noticeable in adults.

Very occasionally, with lumbar disease, a lower motor neurone paralysis is seen from involvement of the cauda equina, the paraplegia then being flaccid. A flaccid paralysis of the muscles innervated by nerves arising at the level of the lesion, as a result of the interruption of function of the anterior horn cells at that level, is sometimes seen, though the muscles supplied from a lower level are spastic.

Thus in low cervical disease the arms may be flaccid while the abdomen and legs are spastic. Localized flaccid paralysis of muscles may also arise from pressure by abscesses upon nerves outside the vertebral canal and is seen not infrequently in the intercostal muscles with thoracic disease. A similar disturbance of sympathetic nerves is sometimes seen¹, giving rise to abdominal distension and other upsets of bowel function, but is by no means confined to paraplegic patients.

Recovery—Type I paraplegia can remain stationary for many months—certainly over a year—and then recover completely. Usually, however, those that recover show some improvement after six months. Recovery is generally associated with—sometimes preceded by—clinical or X-ray evidence that the disease itself is lessening. Pyrexia is less, root pains disappear, or by X-ray bone destruction is seen to have ceased, abscess shadows are smaller, or some recalcification is apparent. Recovery is usually slower than development. Some slight abnormality such as an absent abdominal reflex, an extensor plantar response, or an increase in the knee-jerks, may persist for many months or even years, but on the whole recovery from Type I paraplegia under efficient treatment is, if the patient survives his illness, for all practical purposes complete. Only occasionally does the paralysis of a patient who is under efficient treatment pass from Type I into Type II, remaining permanently after the main activity of the disease is past.

Cause of Death—The direct mortality from Type I paraplegia is difficult to assess. It cannot be said how many patients who die paralysed would anyway have died from their spinal infection. Death with paraplegia may be due, as sometimes in Pott's disease without paralysis, to generalized tuberculosis or meningitis.

Septic absorption from sores and ascending urinary infections are the two causes of death particularly associated with paraplegia. In Type I paraplegia observed by the writer septic absorption from sores has been the most common cause of death (45 per cent), general spread of tuberculosis the next most common (30 per cent).

Cerebrospinal Fluid—Some abnormality is always found in the cerebrospinal fluid in Type I paraplegia. All features of From's syndrome are not always present, but increase of protein is constant.

Xanthochromia and spontaneous coagulation, which depends upon the presence of fibrin, may be absent. In any case the changes are not characteristic of paraplegia from Pott's disease, being the same as those found with syphilitic myelitis or spinal neoplasm.

THE PATHOLOGY AND ETIOLOGY OF TYPE I PARAPLEGIA

Clinically we have seen that Type I paraplegia arises, persists, and recedes in direct relationship to the activity of the disease itself. We therefore expect that the morbid anatomy will reveal the origin of such paraplegia in factors which can appear and later disappear.

The usually accepted explanation of Type I paraplegia is that the cord is compressed mechanically by an 'abscess'. In the majority of fully developed paraplegias of this type an element of compression certainly is present, though the compressing factor, as we shall see, is frequently granulation tissue rather than fluid pus as would be suggested by the term 'abscess'.

Type I paraplegia may, however, occur without true compression of the cord and its meninges. There must therefore be other factors which can alone produce interference with cord function in such cases, and which are worthy of most careful consideration, for such factors may be present also in others in addition to an element of mechanical compression, sharing in the production of the paralysis and providing something more than a straightforward mechanical basis for the paraplegia, which will not therefore necessarily be relieved completely by mechanical means.

The non-mechanical factors thus concerned in Type I paraplegia are the vascular and toxic reactions in the cord secondary to the neighbouring active tuberculosis, and these will be considered before passing on to the more obvious element of mechanical compression.

Type I Paraplegia without Mechanical Compression of the Cord.—The following is a case report of this condition —

Case 1—A boy, A. C., aged 5, was admitted to Pyrford Orthopædic Hospital in May, 1931, with a mid-thoracic kyphos and a history suggesting tuberculous disease for four months. After nine months on a spine frame he developed bronchopneumonia and died. For the last six weeks he had increasing paralysis, and at the time of death he had a spastic paraplegia in extension with sensory loss to the 6th thoracic segment and loss of sphincter control. During life, X-rays suggested that the 5th to the 8th thoracic vertebræ were infected and largely destroyed.

A sagittal section of the spine is shown in *Fig 517*. There is infection of seven vertebræ—not four as was thought during life—and of all intervening discs. It is indeed common—

especially in children—to find at post-mortem that more vertebrae are infected than is shown by X-rays during life. Tuberculous granulation tissue has spread on all surfaces of the vertebral bodies. It forms a large mass anteriorly, and behind—opposite the main area of bone destruction—has invaded the extradural space. Here a rounded mass lies in front of the dura mater and has spread around both sides to form a mass behind the cord also. There is much caseation, and in the central area of destruction is a small abscess containing fluid pus. There is no fluid pus in the extradural space, but here the granulation tissue contains many small areas of caseation. The surrounding tissue reaction is intense. All the soft

parts on the front and sides of the spine are oedematous, including even the fat in the posterior mediastinum. This oedema extends, moreover, for the whole length of the specimen. The extradural space in the central region of the specimen is filled, partly by granulations, partly by oedematous fat and connective tissue. Vascular engorgement is marked, especially below the lesion. The small vessels are distended even on the walls of the aorta. The vessels on the cord itself are congested, especially below the main site of disease. The

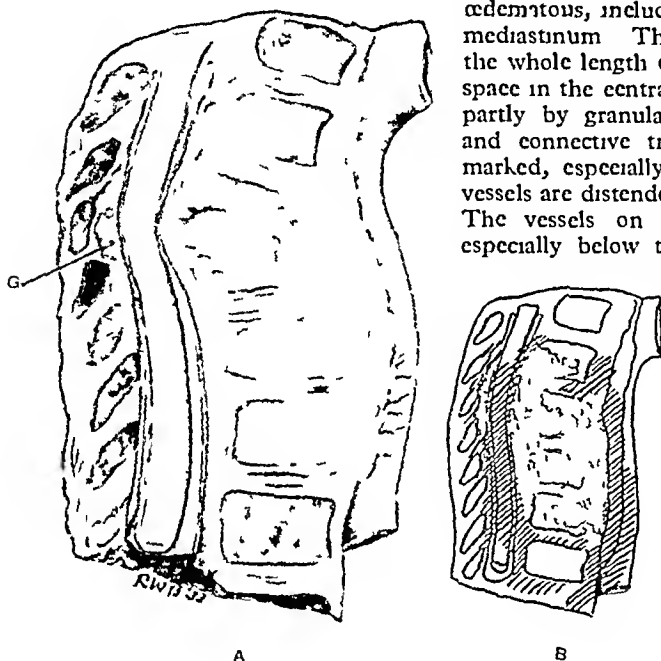


FIG 517—Case 1. Type I paraplegia without true mechanical compression of the cord, with early mid thoracic Pott's disease, in a boy aged 5. Tuberculous granulations have invaded the extradural space and have spread round to the posterior surface of the dura (G). The soft parts of almost the whole specimen are oedematous. In B the area of vascular engorgement (shaded red) around the actual area of tuberculous infection (stippled) is emphasized.

dura mater is intact. Its inner surface is smooth, shining, and normal in appearance.²⁰ Its outer surface is adherent to the epidural granulations, but the membrane itself is not apparently changed macroscopically. With but slight difficulty the granulation tissue can be 'peeled off' the dura mater, leaving the latter apparently normal.

Here, then, is a specimen from a patient with Type I paraplegia in which there is no true mechanical compression of cord tissue. Tuberculosis has invaded the

extradural space and surrounds the dura mater, but the cord itself is not pinched or distorted in any way. What then is the cause of the paraplegia? *A study of specimens from patients with Type I paraplegia reveals only one constant factor—the spread of active tuberculous granulation tissue to the epidural space, where it comes into intimate contact with the meninges.*

Spread of infection to the extradural space may be early, especially with infection commencing in the posterior part of a vertebral body, and is but little checked by the posterior common ligaments, which are wide only behind the intervertebral discs.

Granulations appearing in the anterior part of the extradural space spread round one or both sides of the dura mater and up and down the canal, often finally forming a ring—or rather tube—of infection about the cord. In the granulations small areas of caseation and pus formation appear, but rarely in the early stages of infection is there a collection of fluid pus in the extradural space which truly deserves the term of 'abscess'. The dura mater is found covered with a layer

of shaggy granulations, and at first sight it appears that there is a true tuberculous 'pachymeningitis'. This is the term used to describe this picture in many of the older case reports,^{4, 19} but it is misleading, since the dura mater is not affected by tuberculosis but is acting as an excellent barrier to its spread

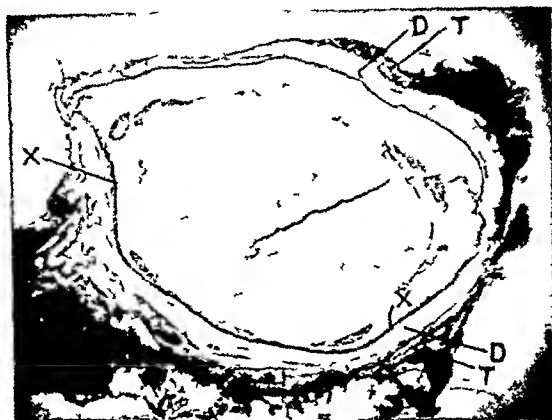


FIG 518—Case BHT 3 Type I paraplegia without compression of the cord. Low-power magnification of a section of the cord and meninges from a man, aged 61, who died in St Thomas's Hospital with a Type I paraplegia of four months' duration with mid-thoracic Pott's disease. The relationship of the extradural granulation tissue (T) to the intact dura mater (D) is shown. Hyperplasia of the dura mater is shown (X X) where thickening is taking place with appearance of fibroblasts and small round cells on its inner surface. The sharp line of demarcation between tuberculous granulations and intact dura mater is everywhere evident.

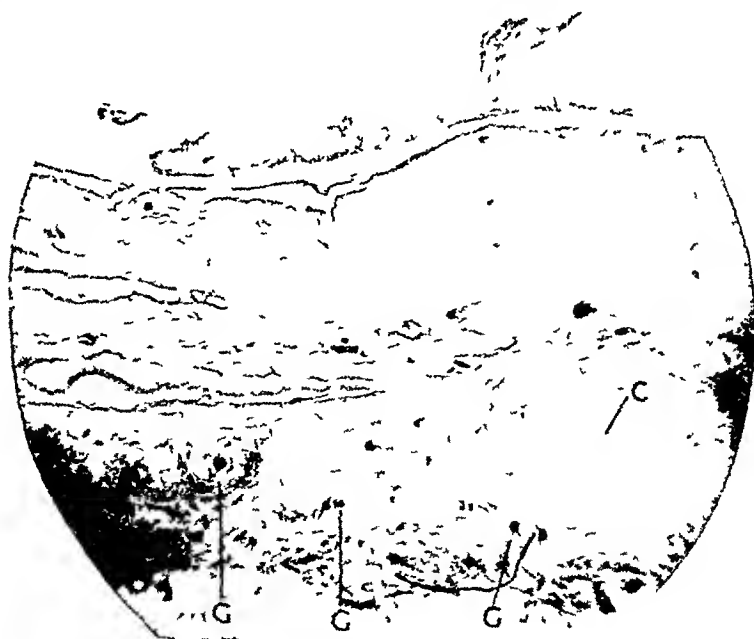


FIG 519—Case BHT 3 Higher magnification than Fig 518 to show the relationship of the tuberculous granulation tissue (below) to the dura mater (above). The two can be easily peeled apart, as to the left of the section. The dura mater shows some thickening and vascular engorgement. In the tuberculous tissue are many giant-cell areas (G) and caseation (C).

It is true, as shown in the photomicrographs Figs 518, 519, that the dura mater may be thickened where it is in contact with granulation tissue, but this is due to the growth of young connective-tissue cells outside the endothelium on the

inner surface, so that the barricade is, as it were, constantly strengthened from within. The correct description is that there is an extradural spread of tuberculosis with some reactionary hyperplasia of the dura mater^{35, 7, 21}. With care the granulations may be stripped from the dura mater, leaving it of normal integrity and appearance.

If, then, paraplegia can occur in the presence of epidural tuberculosis but in the absence of both mechanical compression of the nervous tissue and of infection of the dura mater, we must seek yet other factors as the main cause of the paraplegia. In the above description of a specimen from a patient dying with Type I paraplegia—the wide-spread toxic and vascular-tissue reaction as shown by oedema and vascular engorgement has been stressed—There is nothing unique about this reaction as far as disease of the spine is concerned. It occurs about every active tuberculous focus. It commences as soon as the focus is established, increases—as the disease reaches its height, and disappears with its recovery. It is marked with spinal disease because spinal disease is often a large and, in its early stages, an ill-limited focus of tuberculosis. Once the infection has passed its acute stage and some general and local immunity has been established, this surrounding reaction largely disappears and is comparatively little in evidence about a chronic focus.

In considering the tissue oedema and the vascular engorgement it is hard to say with certainty how far these are interdependent factors. The oedema appears to be distributed all round the lesion equally and is probably largely a local toxic or allergic change.

The vascular engorgement is more definite below the main centre of infection, and is probably due mainly to obstruction of the veins by pressure of the surrounding oedema and of the invading tuberculous tissue (Fig 520). The cord, lying in the centre of the area of intense tissue reaction and separated from it only by the dura mater, undoubtedly shares both in the oedema and the vascular changes. With regard to the latter, it must be remembered that the cord below the uppermost cervical region depends for its circulation upon small segmental vessels which reach it by passing in along the nerve-roots from vessels in the extradural space. These latter, both arteries and



FIG 520.—Type I paraplegia. The cord exposed from behind and the dura opened. The cord in the centre of the figure is lying anteriorly in a bed of tuberculous granulation tissue. The dura mater is intact. Engorgement of all the vessels on the cord, especially below the disease, is well shown. (By permission of Mr H J Seddon)

veins, are connected with branches from the vertebral, ascending cervical, intercostal, lumbar, and ilio-lumbar vessels¹²

Should the vessels of the extradural space be congested, compressed, or destroyed over a wide area, there is no doubt that the circulation in the cord must be greatly affected. A sudden onset of complete paraplegia may in fact occur as a very rare disaster from an acute thrombosis of the vessels in the cord which has spread directly from an extensive thrombosis in extradural vessels. Such a case is described by Seddon (p 774)

There must also be much destruction of the lymphatic vessels, which are numerous in the extradural space and send branches along the nerve-roots which are believed to be connected directly with the intercellular spaces in the cord itself

—Let us now see what microscopic changes are present under these conditions within the cord

In Figs 521-523 we see sections of the cord from two patients both dying with complete paraplegia of

Type I in association with thoracic caries

In both patients there was much extra-

dural tuberculosis, but in neither was there compression of the cord by granulation

tissue or pus

In both the general

histological features are similar

Where the dura mater is in contact

with granulation tissue it is slightly

thickened—partly by œdema,

partly by new connective-tissue

formation on its inner surface

Young connective tissue and

round-celled infiltration are present

about the meningeal vessels

The arachnoid membrane appears nor-

mal and the subarachnoid space is

distinct

In the nerve-roots and

on and in the cord itself there is

some increase of young connective

tissue and round cells about the

vessels, but this must be regarded

simply as a reaction to the



FIG 521—Case B A N 1 Type I paraplegia without compression of the cord. Section of the cord at the level of the disease from a woman aged 58 with early mid-thoracic Pott's disease admitted to the National Hospital, Queen's Square, with a paraplegia for six weeks. Tuberculous granulation tissue had invaded the extradural space, but there was no mechanical compression of the cord. The appearance of fenestration in the large myelinated tracts of the white matter is well shown. (For this and the following illustration the writer is indebted to Dr J G Greenfield, Pathologist to the National Hospital)

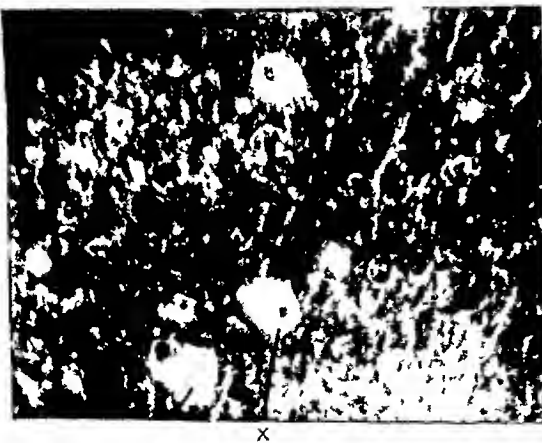


FIG 522—Case B A N 1 Type I paraplegia without compression of the cord. High-power magnification of Fig 521 to show the rounded spaces which give the fenestrated appearance to the white matter of the cord. These appear to be degenerating and distended myelin sheaths, and in many a swollen axis cylinder still persists (X)

extradural changes, for it is very different from anything seen in the characteristic zone of infection outside the dura

In the cord, at the level of the lesion, is one striking change—most noticeable in the white matter. Numerous clear round areas give an appearance of fenestration, most marked in the lateral funiculi, in the anterior fasciculi on either side of the median fissure, and in the bases of the posterior funiculi, as well as all round the periphery. This shows best in the lateral and anterior cerebrospinal fasciculi (the crossed and direct pyramidal tracts). These clear areas are apparently the cross-section of fusiform or rod-like distensions of degenerate myelin sheaths. In

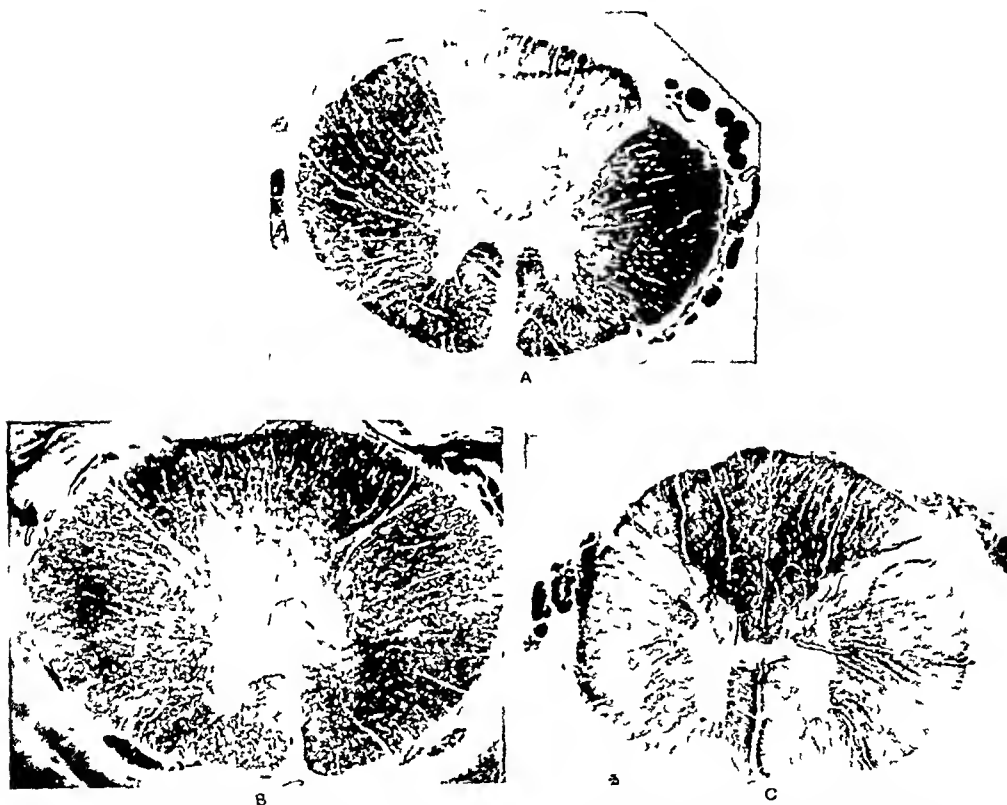


FIG 523—Type I paraplegia. Sections of the cord from a woman aged 43 who died in St Thomas's Hospital with a paraplegia of only six weeks' duration from early mid thoracic Pott's disease. Stained by the Weigert-Pal method to show degeneration of white matter at the level of the disease and in ascending and descending tracts above and below. A, Two nerve roots above the disease; B, At the level of the disease—the appearance of fenestration in the large myelinated tracts is clearly visible; C, Two nerve-roots below the disease. Note that the appearance of fenestration is present only at the level of the lesion.

some of them swollen and degenerating axis cylinders are visible (Fig 522), in others these have disappeared. In a longitudinal section these rod-like spaces appear at intervals in the sheaths, the continuity of the myelin breaking abruptly at either end or fragmenting into poorly staining debris. The exact significance of these distended myelin sheaths is obscure, but, since they occur only at the level of the lesion, they must have some other origin than the simple ascending and descending degeneration of myelin sheaths which extends rapidly above and below (Fig 523). The grey matter shows some degree of perivascular infiltration

and perhaps some unusual preponderance of glial tissue, but the nerve-cells appear little altered

That these changes have arisen as part of the general toxic and vascular reaction about the active tuberculous focus seems certain. Any more exact definition of the changes and of their mode of origin seems impossible.

Charcot⁴ and Michaud²¹ described the cord change as a myelitis—implying that it was actually tuberculous, which it certainly is not. Menard²⁰ considered that it was a "simple œdema", and Zeigler⁴² that it was due to obstruction of afferent vessels. Strumpell³⁵ thought the lesion primarily a degeneration of nerve tissue with a reactionary phagocytic round-celled infiltration and œdema. Schmaus²⁸ described it as a toxic œdema. Fickler⁹ based everything on lymphatic obstruction, and recently Hassim¹⁷ appears also to have favoured this. A very similar appearance with vacuolation in the white matter as described above was produced by Kahler¹⁸ in 1882 by introducing non-infective substances such as beeswax and laminaria into the extradural space, and was discussed by Schmaus.

Whatever other factors, mechanical or otherwise, may contribute to the paraplegia, these toxic and vascular changes in the cord will have to be considered in all cases where the paraplegia is associated with active tuberculosis in the immediate neighbourhood of the cord. They have therefore been described at length, as their importance is undoubted.

It is clear that we have here seen enough cord change to explain such interruption of function. The change, moreover, is of a type which may well allow of much recovery of function when the neighbouring infection—to which it is a response—itself recovers.

The pathological picture agrees with our clinical knowledge of the outcome of Type I paraplegia. The paralysis will generally recover on conservative treatment if the patient lives long enough for his tuberculosis to heal.

Mechanical Compression of the Cord as an Additional Factor in Type I Paraplegia—The occurrence of Type I paraplegia without mechanical compression of the cord has been stressed, since in considerations of treatment the presence of some mechanical factor is so often taken for granted as being present always and present alone.

That some slight pressure is in all cases exerted upon the dura by the granulation tissue, with obliteration wholly or in part of the subarachnoid space, might be thought likely from the differences in the cerebrospinal fluid above and below the focus. That the normal circulation of the cerebrospinal fluid is interrupted is certain from such evidence as Queckenstedt's test and the subarachnoid injection of lipiodol above the lesion, as well as from the chemical changes. The chemical changes are variable, though one—the increase of protein, mainly as globulin—is constant. It is possible, as Greenfield and Carmichael¹⁶ have pointed out, that this last change may be secondary to increased filtration from the vessels from engorgement below the lesion rather than to obstruction to the flow of fluid. Even if the "cavite close" theory of Sicard and Deschamps is true, the obstruction in the subarachnoid space may be due in part at least to hyperplastic thickening of the dura mater and to swelling of the cord itself.

Mechanical compression of the cord is usually present in Type I paraplegia, and such compression may be gross. When compression is by granulation tissue it may be a slight general constriction, the cord being then actually larger than

normal immediately above and below from œdema, or there may be distortion from a mass in front or to one side (*Fig 524*)

When compression is by the fluid pus of an abscess it is frequently very marked, as in *Figs 525, 526*. In the two specimens illustrated certain features are similar. In neither is the bony canal narrowed, in both the epidural space is filled by tuberculous tissue and pus, in both the compressing extradural abscess is but a process of a larger paravertebral abscess pushed back through the main areas of destruction, in both the tension in the tuberculous epidural tissue and in the cord itself must therefore have been the same as that in the paravertebral abscess.

In the writer's experience all specimens showing compression by fluid extradural pus show also that this is an extension from a larger paravertebral abscess. In this the writer agrees with the Sorrels,³¹ but he disagrees with their statement that such connection with a paravertebral abscess acts as a safety-valve, preventing high tension in the part of the abscess immediately related to the cord.

The Sorrels' view is probably correct in cervical and lumbar disease, where a paravertebral abscess can spread easily between muscle planes and sinus formation may result, but in the thorax such abscesses are thick-walled, localized, and fixed, and any surgeon who has drained such an abscess by costotransversectomy knows that its contents are under high tension.

Actually where an epidural abscess is in continuity with an extravertebral abscess within the thorax the tension in the contents must be rising and falling with the respiratory movements, and such changes of tension must be transmitted direct on to the cord like the blows of a water hammer. The extension of a paravertebral abscess to the extradural space must therefore increase the chance of paraplegia on mechanical grounds. Clinical findings corroborate these suggestions, for although almost all patients with thoracic caries develop an extravertebral abscess, sinus formation is rare. It has long been recognized that should a patient with paraplegia from thoracic caries develop a sinus and thus establish drainage naturally the paraplegia will recover rapidly.²⁰ It is of much interest to note that Percivall Pott, writing his first tract upon palsy of the lower limbs in 1779, says that he was particularly led to consider the condition by the case of a boy who had a "restoration of the use of the limbs immediately after a seemingly accidental abscess near the part."

The extramedullary tumour type of paraplegia in which early disease at the back of a vertebral body is not shown by X-rays is clearly a matter of ill chance. Disease has arisen where it can immediately affect the cord.

To summarize the pathology of Type I paraplegia, we may say that it is due to toxic and vascular reaction in the cord, supplemented in many cases by compression by granulation tissue or pus.

Mild and transitory Type I paraplegias are probably due to toxic and vascular cord changes only, without any great element of true compression.

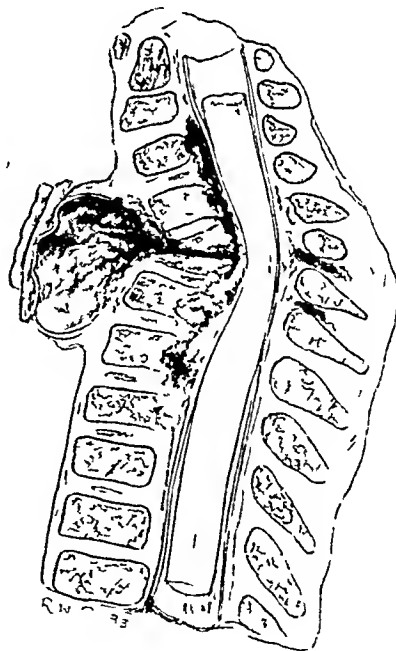
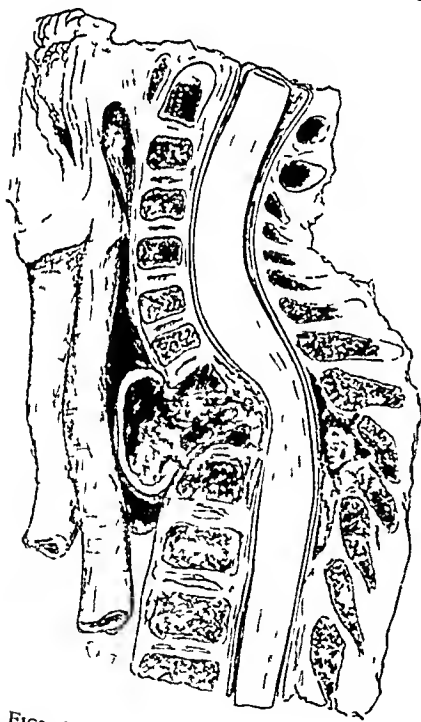
In what percentage of Type I paraplegias the compression factor is gross it is impossible to state. In most of those that die with a severe paralysis it is well marked. Even when it is severe, however, it would seem likely that drainage of pus from a compressing abscess, either spontaneously or as the result of operation, or the rapid recovery of an active tuberculous focus with disappearance of granulation tissue, might well allow of restoration of cord function as long as the compression had not been too long maintained.

PARAPLEGIA IN POTT'S DISEASE

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FIG 524—Case BEH 4 Type I paraplegia with compression of the cord Section of the cord and meninges from a woman aged 52 with severe Type I paraplegia from low thoracic Pott's disease who died in St Thomas's Hospital At post-mortem the extradural space was full of tuberculous granulation tissue and semi-fluid caseous material compressing the cord The cord is seen distorted, and masses of granulation tissue are seen in some areas adhering to the outer surface of the dura mater, which has, however, acted as a barrier to its spread (The fragmentation of the dura mater is an artefact)



Figs 525, 526—Type I paraplegia with true mechanical compression of the cord

Fig 525 shows the upper part of the spine of a child aged 4 The cord is surrounded by granulations and caseous matter which are in continuity anteriorly through the main site of destruction in the upper thoracic region with a thick walled paravertebral abscess containing fluid pus (Specimen No 16 B 2 Museum of the Medical College, University College Hospital)

Fig 526 is another specimen showing a similar state of affairs with cervical disease (Specimen No 153, Museum of Pathology, St Thomas's Hospital)

TYPE II PARAPLEGIA

Type II paraplegia, it will be remembered, was described as paraplegia associated in onset with early active disease but persisting permanently, even when the tuberculous infection in the spine has become completely quiescent. Should the patient survive, the majority of paraplegias with early active disease recover under treatment as the disease itself recovers (Type I). We have seen what pathological factors may produce the paraplegia and yet be capable of resolution to allow of this recovery. A certain small number of paraplegias which have their onset with early active disease remain to a large extent permanently paralysed (Type II).

THE CLINICAL PICTURE

Recognition of the Type—Most paraplegias which finally turn out to be of Type II are at their onset indistinguishable from Type I. It is, however, right to place such cases as soon as they can be recognized in a separate class, since it is clear that some further pathological factors must have appeared to make recovery impossible.

Occasionally a dramatically sudden onset of profound paralysis may suggest the occurrence of some major disaster, mechanical or vascular, and will lead us to expect at the start that the cord will subsequently prove to be irretrievably damaged (see Seddon's article, p 773)

Type II paraplegia is uncommon. Most patients in whom it occurs have had inadequate treatment. This may have taken the form of real neglect, as when a patient refuses proper treatment or is unable to obtain it in the stage of greatest activity of the disease, when bone destruction is occurring rapidly. On the other hand, the patient may be under treatment with good fixation but becomes so ill or develops such severe pressure sores that ideal treatment has to be jettisoned to save life. Most Type II paraplegia is, therefore, the result of a failure to treat Type I paraplegia properly.

As would be expected, since efficient splintage during the destructive stage of the disease has been lacking, Type II paraplegia is almost always associated with great deformity.

Before the stage of permanent paralysis is reached some degree of recovery may occur. Though it may be extensive, the spasticity and motor paralysis is then often somewhat asymmetrical.

Anæsthesia may be patchy and irregular, and control of one or both sphincters may return. This partial recovery has usually occurred at the time when the disease was becoming quiescent, i.e., at the time when Type I paraplegia recovers completely.

In the 'permanent' stage the patient can often live fairly comfortably, getting about with calipers and crutches or sticks, but he is rarely fit for work except in an institution with special accommodation for cripples.

In some, in whom the spinal lesion has been very extensive, infection may grumble on for years, occasionally becoming manifest by some exacerbation, which is then sometimes accompanied by a further deepening of the paralysis. In some, therefore, the paraplegia is associated with continued activity. In some there is no evidence at all of persistence of active disease.

PATHOLOGY AND ETIOLOGY OF TYPE II PARAPLEGIA

In any irrecoverable paraplegia it is clear that at a late stage there will be cord degeneration. What, however, are the factors which lead to this degeneration and which turn a recoverable Type I paraplegia into an irrecoverable Type II?

Firstly, it is certain that many Type II paraplegias are established because disease lasts so long before healing and its effects upon the cord, toxic, vascular, or mechanical, are so profound that the damage to nerve tissue, reparable at first, becomes permanent. Considering the degree of cord change in Type I paraplegia it is surprising that Type II is not more common. This is the paraplegia from long-continued disease.

As a matter of fact, profound Type I paraplegia in flexion, implying involvement of all tracts of the cord, which lasts for more than a very few months rarely recovers. Likewise, a paraplegia which becomes flaccid—though spasticity may return—always appears to be irrecoverable. Fortunately, both are rather uncommon in paraplegia with early active disease. Permanency of paraplegia is not closely dependent upon length of persistence of the paralysis, but rather upon the type and time factor combined. One very rare cause of a sudden onset of irreparable cord damage in early disease is an acute thrombosis spreading into the vessels of the cord from without. An example is described by Seddon.

That a mechanical factor—pressure upon the cord by bone—may sometimes be the underlying factor in Type II paraplegia is made likely by the fact that splintage of the spine has often, for one reason or another, been lacking at the time of most rapid bone destruction, and by the consequent gross deformity which is generally present.

That compression by bone is a common cause of paraplegia has been long disproved, and up to recent times some authorities have even asserted dogmatically that it never occurs. Recently, however, sufficient examples have been described in detail to convince even the most hardened theorists that compression by bone is occasionally present.

The most terrible kyphos may occur without paraplegia. As long as the posterior vertebral arches with the articular processes and posterior intervertebral joints are intact, destruction of the bodies with a falling together in front results only in a hinging of the vertebræ, one upon another, behind. There is a shortening of the vertebral canal but by no means necessarily any loss in its diameter, and a sliding apart of the laminae may actually increase the capacity of the canal posteriorly. Compression of the cord by bone may, however, occur in Pott's disease in two ways—

1 If the posterior intervertebral joints or the pedicles and articular processes are destroyed, a pathological dislocation of the spine may take place with pinching of the cord.

2 If destruction of the vertebral bodies leads not only to the removal of bone but to the formation of loose sequestra or masses of debris, these may be forced back by the collapse of the other bodies into the vertebral canal, where they compress the cord.

Pathological Dislocation—This is comparatively common in suboccipital and atlo-axoid disease, where the posterior intervertebral joints are involved early and massive loss of bone is characteristic. Dislocation in this region is a classical

cause of sudden death in Pott's disease (*Fig 527*). Dislocation at a lower level is uncommon. In the thorax, following destruction of the posterior intervertebral joints, the upper spinal segment either falls down anteriorly in front of the lower, in which case the cord is pinched over the upper end of the lower segment (*Fig*

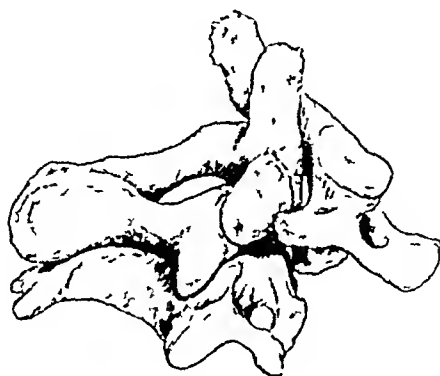


FIG 527—Atlo axoid dislocation. Showing the element of rotation usually combined with the antero posterior dislocation at this level. Massive destruction of the posterior arch of the atlas is also typical (*Specimen No 3868 1, Museum of the Royal College of Surgeons*)

528, A), or slides downwards and backwards over the lower segment, in which case the cord is pinched by the lower end of the upper segment (*Fig 528, B*)

Two examples of dislocation in which the upper segment has fallen downwards and forwards over the lower are shown in *Figs 529-532*. The first

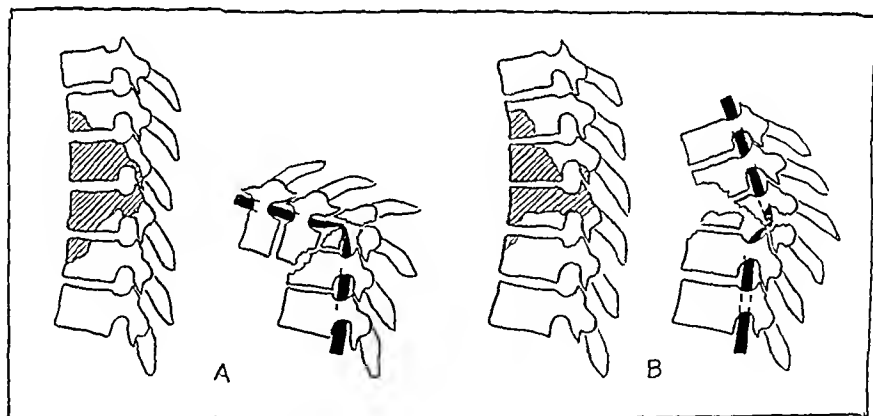


FIG 528—Diagrams showing the two ways in which pinching of the cord may result from pathological dislocation from Pott's disease in the thoracic region. The shaded areas are those in which bone is destroyed. A, The upper segment of the spine has slipped downwards and forwards (as in *Figs 529, 530*), B, The segment has slipped downwards and backwards (as in *Fig 533*)

(*Figs 529, 530*) is from a man aged 33 under the care of Dr B Armstrong at the Royal Sea Bathing Hospital, Margate. The case is reported by Seddon (*Case S 90*). He developed a paraplegia while under treatment for sacro-iliac tuberculosis. This became complete, and he died thirteen months after the onset from generalized



FIG 529

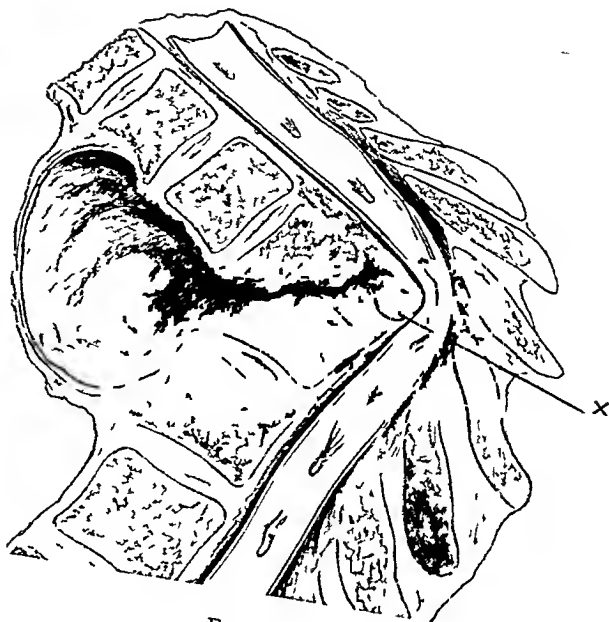


FIG 531



FIG 530



FIG 532

Figs 529-532—Two specimens showing pathological dislocation of the thoracic spine in Pott's disease, with pinching of the cord. In each case the upper segment of the spine has fallen downwards and forwards over the lower, so that the bone compressing the cord is the persisting postero-inferior corner of a vertebral body which has remained in position, forming the apex of the lower segment. Figs 529, 530 are from a case (Case S 90) reported by Seddon to whom the writer is indebted for the illustration. The laminae and pedicles were removed during dissection of the specimen. Figs 531, 532 are from Specimen No 155 in the Museum of Pathology, St Thomas's Hospital.

tuberculosis and sepsis from sores During life caries of the 5th to the 7th thoracic vertebræ was recognized, and by X-rays the body of the 6th thoracic vertebra appeared to project into the spinal canal The second specimen (*Figs 531, 532*) is from the Museum of Pathology at St Thomas's Hospital The presence of a large abscess cavity and of much tuberculous tissue shows that the disease was early and active at the time of the dislocation, but details of the case history are unfortunately lacking

The example of dislocation in which the upper segment has slid downwards and backwards over the lower, shown in *Fig 533*, is from a boy aged 16 under the care of Mr St J D Buxton The case is reported by Seddon (*Case S 62*)

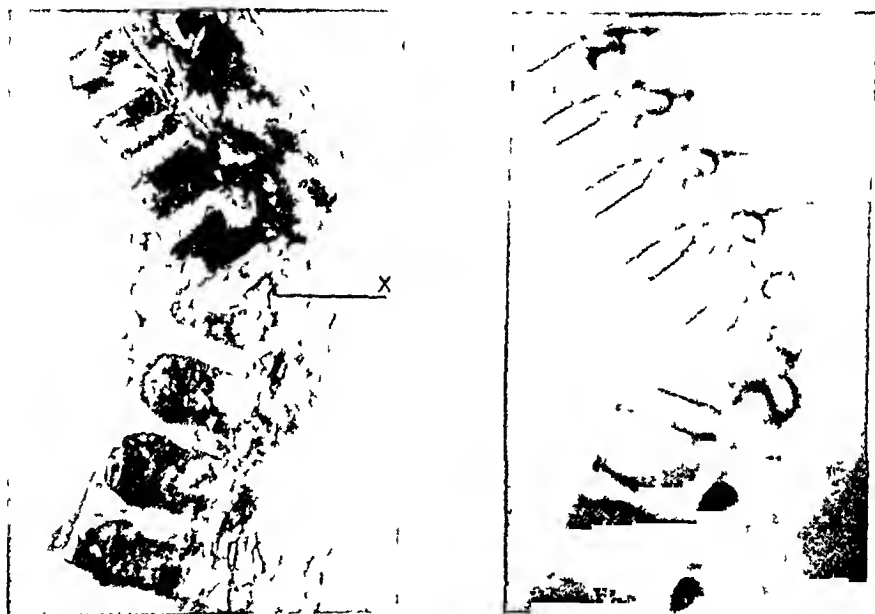


FIG 533 —Pathological dislocation of the spine in Pott's disease, with pinching of the cord The upper segment of the spine has slipped downwards and backwards on the lower The bone compressing the spine is the persisting postero superior fragment of a vertebral body which has remained in position as the lowest point of the upper segment From a patient (*Case S 62*) reported by Seddon, to whom the writer is indebted for the illustration

Mid-dorsal caries was recognized in October, 1928, and paraplegia commenced in November of the same year This became complete, and in January, 1929, he was transferred from a sanatorium to King's College Hospital Laminectomy was performed, extradural abscesses were emptied, and the extradural space was drained for three days He died in April, 1929, from generalized tuberculosis and sepsis from sores, without recovery of the paraplegia In all these three patients the mechanism of the pinching of the cord by the pathological dislocation is made clear by the specimens and X-rays

While discussing compression of the cord from pathological dislocation it is worth while remembering that simple laminectomy for a Type I paraplegia has occasionally been followed by a sudden increase in the depth of the paralysis, which has remained permanently The most probable explanation is that removal of the

only remaining stable part of the spine has allowed dislocation to occur. The writer has seen two such cases, and others are quoted by Seddon.

Pressure by Loose Sequestra or Masses of Débris.—That compression of the cord by sequestra occasionally occurs is well recognized and has been described by Sorrel³¹ and others. The mechanism of the introduction of loose sequestra into the vertebral canal by the crushing down of the bodies in front is easy to understand.

A specimen illustrating this disaster is shown in *Fig 534*. This specimen is preserved in the museum of the Hôpital Franco-Américaine at Berck-sur-Mer and is shown by courtesy of Dr Jacques Calvé. The patient was the victim of a paraplegia which in spite of treatment was complete and showed no signs of recovery.



FIG 534.—A, Specimen showing compression of the cord by displaced sequestra in the Museum of the Hôpital Franco-Américaine, Berck-sur-Mer; B, Diagram of manner of displacement; C, X-ray of specimen, showing the sequestra (S). (By permission of Dr Jacques Calvé.)

At the post-mortem examination it was found that two loose sequestra had been pushed back into the vertebral canal and were compressing the cord.

One rare cause of a very sudden and profound paraplegia coming on with early active disease is what may well be called 'concertina collapse' of a vertebral body. In this an infected vertebra suddenly gives way with expulsion of the bone debris, tuberculous granulation tissue, and pus into the vertebral canal as a semi-solid mass, so that the cord is suddenly and violently compressed. It would seem correct to class this as Type II paraplegia, for it is unlikely that it could ever recover. No example of 'concertina collapse' occurred in the combined series of 193 paraplegias on which this paper is based, and the condition would appear to be rare. A specimen illustrating this has, however, since come into the possession of the writer and is described by Seddon in his paper (p 773). The condition has also been described by Girdlestone.¹³

While discussing compression by bone it is as well to point out a fallacy which

may arise in the interpretation of some specimens. It is not uncommon, especially in irrecoverable late onset paraplegia, to find a flattened and attenuated cord stretched over a consolidated mass of debris which forms the anterior wall of the vertebral canal at the site of angulation. At first sight degeneration of the cord would appear to have followed compression by this mass. Actually the vertebral canal is often quite spacious behind the cord. The bony canal has been shortened by the angulation. Were the cord of normal length and elastic consistency it would lie free and uncompressed in the undiminished canal (as in *Fig 538*)

There is no doubt that in such cases the primary factor is cord atrophy (the cord has had to take on more than it can stand), and that with atrophy the cord has become shrunken so that it is drawn forward by its loss in length to lie in close apposition with the anterior wall of the canal. Such a conception is of importance in interpretation of the pathological picture with a view to considerations of treatment of similar cases.

Clinically it may be difficult to recognize true compression by bone. X-rays taken during life frequently fail to demonstrate the presence of sequestra, and may be hard to interpret as to the presence or absence of minor degrees of dislocation. X-rays following subarachnoid injection of lipiodol are disappointing in differentiating the exact cause of the paralysis. The lipiodol may be held up with gross kyphos formation even without paraplegia, and it is just in those, the cause of which cannot be told clinically, that such injection seems to give the most variable shadows and therefore the least exact information.³² Permanent compression of the cord by annular constricting bands or masses of fibrous scar tissue from healed tuberculosis has been described, but has not been met with by Seddon or the writer

TYPE III LATE ONSET PARAPLEGIA

Type III is late onset paraplegia, which may appear at any time up to many years after the apparent quiescence of the disease.

THE CLINICAL PICTURE

The outstanding characteristics of Type III paraplegia are the late and gradual onset, the incompleteness of the paralysis, and the high percentage of permanent paralysis in comparison to Type I.

Time of Onset—Very variable, a few months to many years after apparent quiescence of the disease. The writer has seen one patient in whom the onset was forty years after her disease was 'cured'.

Development—Always more gradual than Type I. Muscular spasticity usually the first sign. Sensory change may even be lacking throughout, but there is usually widespread but incomplete anaesthesia. Seldom are the lower limbs completely devoid of slight voluntary power, even at the height of the paralysis. Loss of sphincter control is inconstant. Frequently there is loss of bladder control though the bowel is normal. Occasionally complete double incontinence is seen. Vascular and trophic changes in the skin are very uncommon. Even at its height late onset paraplegia is typically incomplete.

Progress and Response to Treatment—Late onset paraplegia is not always permanent as suggested by the Sorrels. Under treatment by fixation in recumbency as for active tuberculosis of the spine some recover completely and many

improve, though it is true that the prognosis as far as recovery of the paralysis is concerned is not as good by any means as in Type I Recovery of late onset paraplegia appears to be more likely in the younger patients

The combined 96 cases of late onset paraplegia of Seddon and the writer show recovery on conservative treatment in 74 per cent of patients under 16 years old, whereas only 52 per cent recovered over that age

If recovery of late onset paraplegia sometimes occurs with conservative treatment as for active tuberculosis of the spine, it would seem likely that such activity is present in these patients and, as in Type I, is the causative factor in the paralysis. If this were so, then we would look carefully for signs of such activity, since we would hope that by treating the spinal disease we would be able to cure the paralysis. Actually clinical evidence of activity of the disease in the spine is generally hard to obtain in late onset paraplegia, but this does not mean that active disease is absent. The real difficulty may be summed up by saying that recurrent or chronic tuberculosis of bones and joints may produce very little general or local reaction, while bone destruction and fluid abscess formation may be negligible and pain completely absent

Pyrexia may be present, but we must exclude tuberculous or other chronic infections in the chest and renal system. X-rays may be indefinite as evidence of activity in the lesion. Extreme kyphos formation makes the degree of bone fusion very hard to assess, actually it is usually less, especially in children, than the X-ray suggests. Calcified abscess shadows may obscure the lesion, but a shadow suggesting a fluid abscess is valuable evidence of activity when it is present. Progress in bone destruction or kyphos formation since a previous X-ray implies further activity at some time, but not necessarily at the time of examination. Pain at the level of infection is suggestive of activity

Examination of the cerebrospinal fluid in late onset paraplegia is not a very dependable test, since we do not know what factors, apart from active tuberculosis, may or may not be present as the result of the old disease and may produce obstruction in the flow of the fluid. Often the protein in the fluid is not raised to the same degree as in Type I paraplegia. Examination of the fluid in a short series of late onset paraplegias shows that those with the higher figures for the protein content recovered better on treatment than the others, so that the raised protein is perhaps here associated with vascular engorgement from the proximity of active disease rather than with any other factor. The series (see Table, p 760) is, however, too small to be dogmatic in this respect

As a matter of fact clinical evidence of activity of the spinal focus, though most commonly found in those patients who recover, may be present with some late onset paraplegias which remain permanent, and again is sometimes unobtainable in those that recover, so that it is far from reliable in making a prognosis

The only real test of ability to recover is to put the patient under treatment by fixation in recumbency as for active (persistent or recurrent) tuberculosis of the spine and await results, whether such disease is clinically present or not. With the passage of time Type III paraplegia is then found to fall into one or other of two subdivisions—

Type IIIa—Late onset paraplegia recovering on conservative treatment as for active tuberculosis of the spine *Recovery starts within 9 months*

Type IIIb—Late onset paraplegia not so recovering

Recovery in Type IIIa usually commences under treatment within nine months The writer has, however, seen late onset paraplegia remain stationary under treatment for eighteen months and then recover completely (E C, age 13, Pyrford Orthopaedic Hospital, 1931)

The direct mortality rate from late onset paraplegia is negligible Skin sores do not occur and ascending urinary infection is rare

PROTEIN OF THE CEREBRO-SPINAL FLUID IN LATE ONSET PARAPLEGIA IN
RELATION TO END RESULTS OF TREATMENT

SERIAL NUMBER OF PATIENT	PERSISTENCE OF PARAPLEGIA PRIOR TO ESTIMATION	PROTEIN IN CEREBROSPINAL FLUID*	RESULT OF FURTHER CONSERVATIVE TREATMENT OF THE PARAPLEGIA AS FOR ACTIVE TUBERCULOUS DISEASE OF THE SPINE	
(S) 58	Months 4	G per 100 c c 0.9	Recovered	—
(B) W N	4	0.4	Recovered	—
(B) D S	1	0.3	Recovered	—
(S) 68	1	0.24	Recovered	—
(B) E C	3	0.15	Recovered	—
(B) J B	12	0.1	Recovered	—
(S) 99	10	0.1	—	No Recovery
(S) 22	2	0.08	Recovered	—
(B) J D	10	0.06	—	No Recovery
(B) E B	6	0.05	—	No Recovery
(S) 97	2	0.03	—	No Recovery
(S) 32	24	0.015	—	No Recovery

* Normal 0.01-0.03

THE PATHOLOGY AND ETIOLOGY OF TYPE III PARAPLEGIA

Inquiry into the pathology of Type III paraplegia is hampered by the difficulty of obtaining specimens of the spine from such patients, since they seldom die in hospital. Certain specimens are, however, available which prove, as we have suspected on clinical grounds, that the cause of the paralysis may be activity of the old tuberculous disease in the spine. It is desirable to emphasize that such activity is in whole or part of the old bone lesion where it abuts upon the cord. It is no new process such as a tuberculous pachymeningitis, a term often used in the past to explain away any form of paraplegia in Pott's disease to which no definite origin could be assigned.

Case (B) W N, Addenbrooke's Hospital, Cambridge. A man, aged 52, was seen in June, 1933, with a late onset paraplegia of five months' duration. Both legs were markedly spastic, but there was some voluntary power in all muscle groups except the dorsiflexors of the toes and feet. There was patchy anaesthesia up to the 2nd thoracic segment, most complete below the knees, and partial urinary incontinence. He had a marked kyphos at the cervico-thoracic junction, which had been present unchanged for twelve years. When this

kyphos had first appeared he had had a good deal of pain, both root pain and locally at the site of disease. He had never received any treatment for this, having managed to keep on at his work as a farm labourer, and for ten years since had had no symptoms. At the time of examination he had also an early active focus of tuberculosis in the low lumbar spine, but clearly this was in no way associated with the paralysis. Clinically and by X-ray the old cervico-thoracic lesion appeared in all probability quiescent.

He was rested in recumbency, and for sixteen weeks showed steady improvement. Voluntary power returned to all muscle groups in the legs, and anaesthesia was present only below the knees and in one area on the abdominal wall. Bladder control became normal. The upper abdominal reflexes, previously absent, reappeared, but the lower were still absent.

On Oct. 27, 1933, he had a thrombosis of the right iliac vein following pressure by a retroperitoneal abscess arising in connection with the lumbar disease. He died the next day after pulmonary embolism.

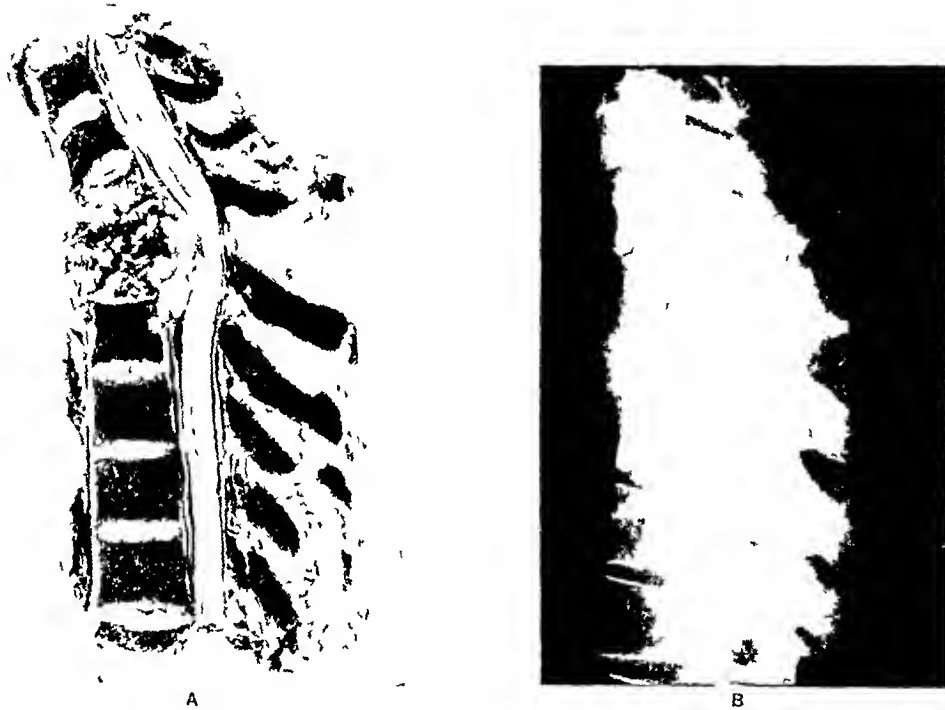


Fig. 535—Case (B) W N. A, Specimen of spine from a patient with late onset paraplegia Type IIIa. Active disease is present in a chronic form. The cord is compressed by a mass of granulation tissue and caseous material containing many areas of calcification. B, X-ray of specimen. (Author's specimen, Addenbrooke's Hospital, Cambridge.)

Examination of the spine of this patient (Fig. 535) showed re-activity of the old cervico-thoracic lesion, with many of the main features which might be expected in paraplegia of Type I with early active disease. Tuberculous granulation tissue and caseous material occupied the extradural space, the cord being compressed thereby against the laminae. At the site of the main bone destruction and in front of this was an abscess containing caseous material.

The most remarkable feature of this case seems to be that in spite of such gross changes the paraplegia was recovering steadily, and, indeed, for a Type III paraplegia, rather rapidly. In contrast, however, with the appearances in early active disease the granulation tissue and caseous matter were semi-calcified. Moreover, the surrounding vascular engorgement and oedema so characteristic of early disease were almost entirely absent.

On microscopy the dura mater was found to be forming an efficient barrier to the spread of tuberculosis and was marked off sharply from the granulation tissue without, but in its outer layers showed rather more round-celled infiltration than is usual in similar sections with early active disease. Sections of the cord showed round-celled infiltration about the vessels, and some degree of vacuolation in the white matter. The appearances were hard to interpret exactly, but on the



FIG 536—Case (B) W N. Sections from same specimen as Fig 535. A, Section showing both active and calcified tuberculous tissue lying against the intact dura mater. (X) Area shown in B. (Y) Area shown in C. B, Section showing, above, the thickened and sclerotic but intact dura mater, and below, tuberculous tissue containing active areas with well-marked giant-cell systems and many deposits of calcium (Ca). C, Part of the cord showing considerable atrophy of nerve cells and fibres and perivascular cellular infiltration and fibrosis.

whole there would appear to be less vascular engorgement and vacuolation both in the grey and white matter, and more permanent atrophy with disappearance of nerve-cells and fibres, than in paraplegia of Type I (Fig 536).

The clinical differences between this paraplegia and that of paraplegia of Type I must in fact have been dependent upon the relative inactivity of this chronic tuberculous focus as opposed to a similar focus at an early active stage. *This chronicity, with the consequent lack of surrounding toxic and vascular change,*

seems to be the essential point to be appreciated in this case The actual mechanical pressure upon the cord had no doubt arisen very slowly, and, as Gowers¹⁵ has pointed out, the effect of pressure upon the cord is directly proportionate to the rapidity with which it comes about

Another specimen of great interest is one preserved in the Pathology Museum of the Medical School at University College Hospital, which shows the condition of the spine and spinal cord in a girl who died from intercurrent disease following recovery several years earlier from a late onset paraplegia—(*Specimen No 16 B S*) The specimen (*Fig 537*) is from a girl treated many years ago at University College Hospital She was first seen with a thoracic kyphos at the age of 6, but as the disease was not considered active was allowed to remain at home In the next three

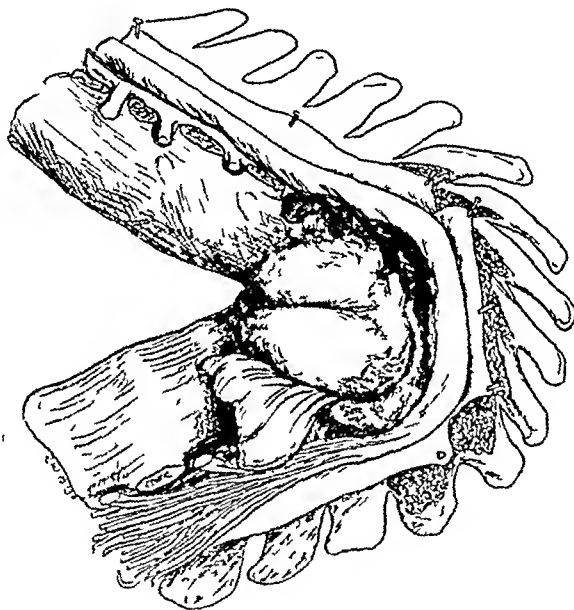


FIG 537—Specimen showing the spine of a girl who died from intercurrent disease six years after recovery from a late onset paraplegia of Type IIIa There is no mechanical constriction of the cord Anteriorly the cord is lying against masses of calcified debris and old tuberculous material, and in front and on both sides of this are large paravertebral abscesses Case report and description in text (and in *Illustrated Medical News*, 1889, cxciii)

or four years the deformity increased and plaster jackets were applied In the light of our present knowledge of thoracic Pott's disease in a child there is no doubt that the disease was active during these years Six years after being first seen—at the age of 12—she developed a widespread late onset paraplegia from which, however, she recovered on treatment in hospital For a further six years she remained in good health At the age of 18 she died of bronchitis

It is clear from the specimen that even at the time of death there was no assurance that the tuberculosis of the spine was permanently healed Big semi-calcified paravertebral abscesses and masses of debris lie in the concavity of the kyphos in close relation to the cord These form a site in which live organisms might lie dormant for years, finally becoming active again and being once more

the cause of reactionary changes in the cord. In this specimen there is no evidence that there had ever been true compression of the cord, which is of apparently normal calibre and has accommodated itself well to changes in the vertebral canal, though the latter is angulated at two points by more than sixty degrees. If any compression was present at the time of the paralysis, it has disappeared spontaneously. There seems to be no alternative to the suggestion that the late onset paraplegia in this girl was associated with persistent or renewed activity of the spinal lesion.

When we consider the clinical findings in conjunction with such specimens as the above it seems certain that the vast majority at least of late onset paraplegias which recover under treatment (Type IIIa) are due to such persistence or reactivity of the spinal disease, and since Type IIIa and Type IIIb paraplegias are usually indistinguishable at their onset, the relevance of this with regard to treatment of late onset paraplegia as a whole is obvious.

It seems likely that an unbalanced deformity of the spine at the end of treatment for the original disease may be of great importance in keeping up or in precipitating activity of the disease, especially in a child, in whom bone fusion may be long delayed.

The weight of the head and shoulders falling in front of the old focus must exert upon it a constant levering action and must undoubtedly tend to maintain a state of strain, with reaction in the bones and soft parts, which can but favour further activity of the disease (see Seddon's article, p. 793).

What now of those patients with late onset paraplegia who fail to recover on conservative treatment—Type IIIb? Clinically there may be evidence that disease of the spine is active in these patients at the time of onset of the paralysis, just as there is at times in Type IIIa. It is probable that much Type IIIb paralysis is at its onset of Type IIIa, and that just as in Type I some factor may be added which makes recovery of the cord impossible and turns it into Type II, so in these patients cord degeneration becomes established for one reason or another as time goes on.

The simplest explanation is that Type IIIb paraplegia is the result of a slowly progressive avascular degeneration arising from repeated minor circulatory damage by chronic disease grumbling on nearly year after year with very little outward sign.

How far pressure upon the cord by bone or hard debris is sometimes the primary cause of late onset paraplegia is hard to assess. Sometimes it may be a primary factor. At any rate, true compression by bone has been found at operation and after death in late onset paraplegia, and Seddon describes such cases, but such compression would not have arisen without reactive disease which allowed of further bone destruction, and it is the occurrence of this reactive disease which requires emphasis. As mentioned when discussing Type II paraplegia, gross primary compression by bone is probably always associated with rapid destruction, and may be expected to occur therefore almost entirely with early disease. Frequently at post-mortem examination of a patient with irrecoverable late onset paraplegia the cord is found stretched over a mass or bar of bone in the anterior wall of the canal, but it must be remembered that once some degree of cord atrophy is established the cord will lose in length and elasticity, and will therefore be drawn forward to lie against the anterior wall of the canal, which, at the best, will consist of rough calcified debris and scar tissue and may perhaps be still the site of a low-grade infection. Further embarrassment of the cord by mechanical irritation will

then occur and a vicious circle will be established, still further cord degeneration following

—With late onset paraplegia there is sometimes a history of a recent increase in the kyphos. With most this is doubtless a sign of active disease with some further destruction of bone — with some, especially with children in whom growth is occurring, it may at times be simply the result of faulty mechanics in an unbalanced spine. In any case, an increasing kyphos will add to the possibility of the pushing back of debris towards the cord, and this, even if it produces no gross compression, will add to the likelihood of an irritation factor being concerned in the production of cord degeneration

The following case of Type IIIb paraplegia provides some interesting evidence in respect to the above possibilities

Case (B) E S 81 —A girl, aged 3, was admitted to Queen Mary's Hospital, Carshalton, under Dr Pugh, with early mid-thoracic Pott's disease, and underwent two years' conservative treatment. Thereafter she was at home for four years in perfect health. At the age of 9 she developed a partial, but steadily deepening, paraplegia. She was readmitted to hospital with an incomplete motor and sensory paraplegia with double incontinence.

Under conservative treatment on a frame she made considerable progress at first, but later the paralysis again deepened and became stationary. After three years, being now aged 12, she had very little voluntary power in the lower limbs except in the dorsiflexors and plantar flexors of the feet and toes.

Sensory loss was patchy to the mid thighs on both sides, and sphincter control was imperfect. It was decided to perform a laminectomy in the hope of relieving the cord from some mechanical pressure. At operation the cord was found attenuated and flattened lying against the front wall of the canal. Posteriorly there was plenty of room in the extradural space and there was no evidence of narrowing of the spinal canal as a whole. The girl died of post-operative shock.

Examination of the specimen of the spine and cord (*Fig 538*) shows that the disease had evidently become quiescent in the last years before death. At first sight there would appear to be compression of the cord by a bony mass pushed back from the main area of disease where the remains of three vertebral bodies are fused into a solid mass.

The bony canal has been much shortened by the angulation so that, unless the cord were even more shortened so that it is compelled to take the shortest possible course against the anterior wall of the canal, it would be lying in the posterior part of the canal where there is room to accommodate it. The relationship of the bony walls of the canal to the cord are not easy to make out in the drawing of the specimen, as destruction of the laminae at operation obscures the true relationships, but the accompanying diagram makes this clear (*Fig 538 B*). The dura mater is quite unattached to the wall of the canal anteriorly. Microscopy of the cord (*Fig 539*) shows general atrophy, with complete loss of differentiation of grey and white matter—though a fair number of medullated fibres are still present. Blood-vessels in the cord itself and in the meninges are scanty and show perivascular fibrosis. The subarachnoid space is crossed by a few fine adhesions. The dura mater is somewhat thickened, especially antero-laterally, and both in the dura mater and in the region without, which was probably at one time invaded by tuberculous granulation tissue, there are deposits of calcium. This specimen is interpreted, therefore, as showing primary cord atrophy rather than atrophy secondary to compression by bone.



FIG 538—Case (B) E S 81. A, Specimen from a girl aged 12 with an irrecoverable late onset paraplegia Type IIIb. Laminectomy has been performed. The atrophic and shrunken cord is seen lying against the fused mass of bone forming the anterior wall of the canal. B, Diagram to show relationship of walls of vertebral canal to the cord. C, X ray taken shortly before death. The specimen is at Queen Mary's Hospital, Carshalton.

In no specimens of late onset paraplegia, any more than in paraplegia with early active disease, has the writer seen any evidence of chronic tuberculous pachymeningitis as the cause of the paralysis



FIG 539—Case (B) E S 81. Half section of the cord at the level of the old disease from the specimen shown in Fig 538. C, Atrophic cord without differentiation between white and grey matter. A fair number of intact axons are however present. Very few nerve cells are visible. D, Somewhat thickened dura mater showing many deposits of calcium (Ca).

SUMMARY

A survey has been made of a series of 186 patients with paraplegia in Pott's disease

From a clinical study of these and from an investigation of the post-mortem pathology it is suggested that paraplegia in Pott's disease falls into certain clinical types with which are associated constant etiological factors

Early Onset Paraplegia.—

Type I—Paraplegia with early active disease, arising, persisting, and diminishing in direct relationship to the activity of the tuberculous infection of the spine. The only constant etiological factor is the presence of active tuberculosis in immediate relationship to the dura mater. In all such the paralysis is associated with much toxic and vascular reaction in the cord, and in some this alone is the cause of the paraplegia. The importance of this reaction in all paralyzes where active disease is present is emphasized. In many the additional factor of true compression of the cord by granulation tissue or pus is also present.

Type II—Paraplegia associated in onset with early active disease but persisting

on permanently even if tuberculous infection in the spine has become completely quiescent. The causative factors in the early stages are usually the same as in Type I. In the permanent stage there is in many simply a varying degree of cord atrophy from reaction to too long continued activity of the disease before quiescence is attained. In others there is true compression of the cord by bone or debris. Compression by bone can only result from pathological dislocation of the spine or the displacement of free sequestra.

Late Onset Paraplegia —

Type III—Late onset paraplegia appearing at any time, up to many years, after apparent quiescence of the disease. Emphasis is laid on the fact that most such paraplegias are associated with low-grade activity, persistent or recurrent, of the spinal infection.

In some (Type IIIa) the causative factors are the same as in Type I, but modified, just as the disease itself is modified. In many such the paralysis will recover with conservative treatment. In others (Type IIIb) the main causative factor is avascular cord degeneration from too long persistent reaction to the disease.

True primary cord compression by bone, though it may occur, has comparatively small place in the causation of late-onset paraplegia, though apparent compression, which is due to primary atrophy, with shrinking of the cord so that it comes to lie later against the anterior wall of the canal, is common.

Tuberculous pachymeningitis, often mentioned as a probable cause of paraplegia, has not been encountered.

To save repetition, a bibliography of the subject, and an acknowledgement of the writer's indebtedness to those who have been of assistance to him in this work, are given in conjunction with Seddon at the end of the next paper.

POTT'S PARAPLEGIA: PROGNOSIS AND TREATMENT

BY H J SEDDON

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PART I THE CLINICAL CLASSIFICATION OF CASES OF POTT'S PARAPLEGIA IN RELATION TO PROGNOSIS

IN any disease the practical value of a clinical-pathological classification is proportionate to the reliability of the guidance it gives in regard both to prognosis and treatment, and this is particularly true of Pott's paraplegia

The only serious attempt hitherto to deal with Pott's paraplegia along these lines has been made by Mme Sorrel-Dejerine, whose weighty monograph³⁰ has won acceptance in many places and raised storms of opposition in others. It is sufficient to say that Butler and I have been quite unable to fit even the majority of our 186 cases into the attractive "early-abscess-recovery" and "late-pachymeningitis-permanent paralysis" groups which she described—after the study of only 40 cases. Not only did some of our early cases fail to recover, and many of our late cases recover completely, but in the specimens from cases of late paraplegia that we were able to examine there was no evidence whatever of pachymeningitis, the condition which Sorrel-Dejerine holds responsible for most of the late cases of paraplegia. I regret that considerations of space prevent me from appraising her thesis in detail, but the question has already been referred to by Butler and we hope to deal with the matter more fully elsewhere at a later date.

The first part of this paper is an attempt to show that the classification of the various types of Pott's paraplegia agreed on by Butler and myself is a fairly accurate guide to the prognosis in this disease.

It is generally agreed that with good conservative treatment, 60 to 90 per cent of all cases of Pott's paraplegia recover completely. In my series of 100 cases there were 109 attacks of paraplegia. Seven histories are too recent to be of statistical value. In the remaining 102, there were 71 complete and 3 partial recoveries, and 28 patients were permanently paralysed. This gives a recovery rate of about 70 per cent. There were 21 deaths, of which 8 were directly attributable to the paraplegia. There is no doubt that the death-rate in spinal caries is considerably increased by the incidence of paraplegia.³⁸

Must we accept this proportion of permanent or fatal paraplegias as inevitable, and if so, is there any way of recognizing the unfavourable cases in advance? Before attempting to answer this question it is necessary to consider certain clinical signs that are of importance in prognosis, quite apart from the clinical-pathological types to be described later. These signs are common to all forms of Pott's paraplegia.

Age—I am in entire agreement with Sorrel-Dejerine in finding that recovery from paraplegia is not greatly influenced by the age of the patient. In my series the chance of recovery improved slightly as the age increased up to 60 years, but

the two patients who developed paraplegia after this age died within eighteen months without recovering from their paralysis

Paraplegia in Extension—This indicates an incomplete lesion of the cord—that is to say, certain tracts have escaped compression, but although the prognosis in this condition is good, it does not necessarily follow that the damage done is always repairable. Out of 67 patients with paraplegia in extension, 54 recovered completely, 3 recovered incompletely, 2 recovered after several years, and 8 (patients in whom paraplegia in extension changed later to paraplegia in flexion) were permanently paralysed. Painful and excessive spasm of the limbs in paraplegia in extension is not an unfavourable sign

Paraplegia in Flexion—In paraplegia in flexion, which indicates involvement of the whole thickness of the cord, the prognosis is bad. All 12 cases with this condition were permanently paralysed. However, a case of recovery after paraplegia in flexion has been recorded by Souques³³ in a woman aged 51 years, and one of Butler's 15 cases of paraplegia in flexion recovered almost completely.

Flaccid Paralysis—In this the prognosis is equally bad. Although spasticity may return, complete recovery is almost unknown. In 1 case out of 7 the patient recovered a little voluntary power in the lower limbs a few months before he died of amyloid disease. The other 6 patients were permanently paralysed.

Duration of Motor Paralysis—In the past considerable attention has been paid to the duration of paralysis, and as motor changes generally appear long before, and remain for some time after, sensory changes are present, it is the duration of motor paralysis that has generally been studied. I have not found my own or the published statistics of the total duration of paralysis of the slightest value in prognosis. As recovery may sometimes occur after paraplegia lasting for as long as six (Case S 5) or seven (Case S 7) years, a prognosis based on the length of time that paraplegia has been present resolves itself into nothing more than a pious hope that recovery will occur in any case of paraplegia in extension of not more than seven or eight years' duration.

A far more exact prognosis may be made from a study of the duration of total motor paralysis. This must not be confused with complete paraplegia, i.e., paraplegia in flexion. By total motor paralysis is meant paraplegia in extension with complete loss of voluntary power. We already know that complete loss of voluntary power resulting from paraplegia in flexion or flaccid paralysis carries a hopeless prognosis. If voluntary power is never entirely lost, recovery is assured. In my series there were 21 cases in which some degree of voluntary power was always present, every one made a complete recovery, though in one case (Case S 7) it was delayed for seven years. On the other hand, where total motor paralysis is present, the prognosis becomes rapidly worse with each month that elapses after its onset; there were 13 recoveries after three months of total motor paralysis, 11 after six months, 3 complete and 1 incomplete recovery after nine months, 2 after one year, 4 complete and 3 very incomplete recoveries in the second and subsequent years. The chances of complete recovery from total motor paralysis of more than six months' duration are extremely poor

Sensory Changes—A study of the sensory changes is of little value in prognosis. It may be said that as a rule the prognosis is good in cases where sensory changes are absent, but the return of sensation in a case where it has been lost, though encouraging, does not necessarily indicate that complete recovery can be expected.

A number of cases in this series recovered sensation completely and yet the motor paralysis was permanent Sorrel-Dejerine believes that the prognosis is bad in

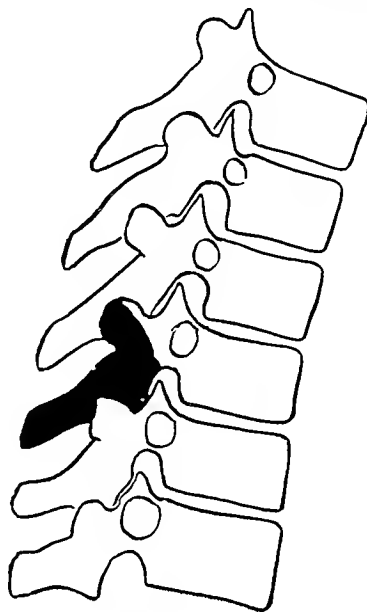
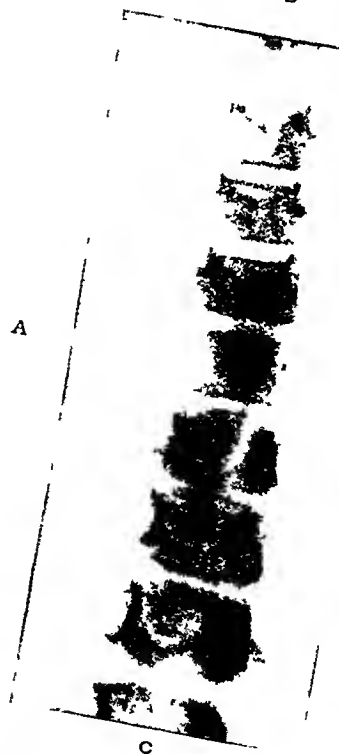


FIG 540—Case S 36 A, Lateral X-ray of the spine of a boy of 9 who was admitted to hospital with mild paraplegia. There is a destructive lesion of the pedicles, laminae, articular processes, and spinous process of Th 8. A typical lesion of the vertebral bodies appeared later, the paraplegia cleared up spontaneously. B, Explanatory drawing of A. C, X-ray, showing lesion at A. (Dr J G Johnstone's case Princess Mary's Hospital, London County Council, Margate)

cases where sensory changes make their appearance many months after the onset of a purely motor paraplegia, this was true in four of my cases, but not in the remaining two.

There is, however, one form of sensation the absence of which is of great prognostic significance. Vibration sense is lost only in cases where compression is so severe that the posterior columns of the cord are involved. When the vibrations of a tuning-fork cannot be felt on the bony points in the lower limbs the prognosis is bad. Recovery from paralysis occurred in only 2 out of 11 patients who had lost vibration sense in the lower limbs. One of the patients who recovered (Case S 36) is of unusual interest. This child suffered from paraplegia as a result of disease involving a thoracic neural arch (Fig 540), and loss of vibration sense was present even when the motor paralysis was well on the way to recovery. Presumably the lesion producing the paraplegia affected the cord from behind forwards.



instead of in the usual way, and derangement of conduction in the posterior columns persisted when the pyramidal tracts had almost recovered

Sphincter Changes—The absence of derangement of function in the bladder and rectum is an encouraging sign. Only 4 of the 33 patients who remained entirely free from sphincter trouble were permanently paralysed. In cases where the bladder and rectum are involved the recovery of normal sphincter action does not always indicate that the paraplegia will clear up completely, in *Case S 19* control returned in six months, and yet this child is permanently paralysed. One of the causes of death in patients with involvement of the bladder is ascending urinary infection, yet only a few fatalities can be attributed to this complication, and it is remarkable how readily severe infection of the urinary tract responds to treatment, even though the paraplegia is permanent. Severe urinary infection occurred in 6 of my cases, but in none was it the cause of death.

Pregnancy—According to Myers²² pregnancy may determine the onset of an attack of paraplegia in a patient with incompletely healed spinal caries. I have seen one patient with spinal caries who had an attack of paraplegia with each of four pregnancies, and in one of Butler's cases (*Case B 7*) the onset of a fatal paraplegia coincided with pregnancy.

THE CLINICAL-PATHOLOGICAL TYPES

The classification proposed by Butler distinguishes three types of Pott's paraplegia —

Type I—Paraplegia with early active disease, arising, persisting, and diminishing in direct relationship to the activity of the tuberculous lesion in the spine.

Type II—Paraplegia associated in onset with early active disease, but persisting indefinitely—even if the tuberculous infection in the spine has become completely quiescent.

Type III—Paraplegia occurring after a tuberculous spine appears to have healed, often in patients who have enjoyed many years of good health and active life.

TYPE I PARAPLEGIA

Type I has been so familiar since the time of Pott that a detailed description of the condition is no longer necessary. Paraplegia may be the first sign of spinal disease, and in adults, particularly, often occurs before any deformity is apparent. Secondly, weakness of the lower limbs may be complained of by a patient who has been aware of trouble in the back for some weeks or months, in this case the history is "back first—then legs". Lastly, paraplegia may develop in cases of active caries receiving the most efficient hospital treatment.

It has been shown by many observers, and again by Butler and myself, that this type of paraplegia is the result of the presence of active tuberculous disease in the immediate vicinity of the cord—the 'abscess' of former writers (see Butler's article, p 744)—and the waxing and waning of activity in this tuberculous focus will determine the course of the paraplegia. Usually the paraplegia is mild at first, then severe—often with sensory disturbances and sphincter involvement, finally, after several months, signs of improvement appear, and at last after a painful and anxious ordeal, the patient recovers full control of his lower

limbs In a few cases the course of the paralysis is less severe, and never amounts to more than a few weeks or months of spasticity and weakness of the lower limbs

Every case conforming at first to this type will recover unless certain things happen —

1 The initial infection may be so virulent that the patient dies rapidly of tuberculosis before the paraplegia has run its course. We are all familiar with these tragic cases of overwhelming tuberculous disease in which the spinal lesion is merely incidental, the most that can be said of the paraplegia is that it hastens the end

2 The disease persists for so long before finally healing that the damage to the cord, reparable at first, ultimately becomes permanent. Activity has persisted for longer than the cord can tolerate

3 An 'accident' occurs, vascular or skeletal, resulting in permanent damage to the cord

TYPE II PARAPLEGIA

Type II comprises the second and third contingencies just described. The Type II case is therefore generally a Type I case 'gone wrong', however, it is sometimes a Type II from the first, without any chance of recovery from the paralysis even in the earliest days. Fortunately, it is often possible to recognize the Type II case in advance, which is of value in prognosis and treatment, there are certain danger signals that herald the Type II paraplegia, and they must constantly be watched for

1 PARAPLEGIA OF SUDDEN ONSET RAPIDLY BECOMING COMPLETE

Two conditions are known to be responsible for this syndrome, and in both the prognosis is bad

'Concertina' Collapse of a Vertebral Body.—A good example of this condition is described by Girdlestone¹³ (Case 4), which is typical of those recorded by other observers

A patient, aged 21 years, was admitted to hospital with backache of two weeks', and paralysis of the legs of six weeks', duration. On admission there was complete flaccid paralysis of the lower limbs, loss of all forms of sensation below the pelvis, and double incontinence. X-ray examination showed collapse of the body of Th 11 with almost normal intervertebral discs above and below. Laminectomy was performed immediately, "after full exposure, flattened area opposite D 11 filled out, but pulsation never seemed as full as at higher level. There was a prominence of the theca opposite D 11. An aneurysm needle passed in front of theca at this level failed to find debris"

The operation was not followed by any immediate improvement. Some degree of recovery occurred ultimately, but spasticity of the lower limbs and incomplete sphincter control persisted

The cardinal signs of concertina collapse are total paraplegia of early and rapid onset, and the radiographic demonstration of collapse of a tuberculous vertebral body

A case of Butler's is of unusual interest in that paraplegia due to concertina collapse was superimposed on a Type I paraplegia

A boy of 19 was admitted in November, 1932, to the Shropshire Orthopaedic Hospital, Oswestry, under the care of Mr Harry Platt. There was a history of three months' weakness in the lower limbs. On admission the patient was found to have a Type I paraplegia due to a lesion at the thoraco-lumbar junction

Early March, 1933 —X-ray showed narrowing of the space between Th 8 and 9 ? second focus of disease

Late March, 1933 —Sudden deepening of paraplegia, level of sensory disturbance rose to Th 8, voluntary power entirely absent, loss of sphincter control X rays showed partial collapse of Th 8 with destruction of pedicles and transverse processes



FIG 541 — Concertina collapse of the eighth thoracic vertebral body. Diagnosis confirmed histologically. (Reproduced by kind permission of Mr R W Butler)

April 22, 1933 —Laminectomy by Mr Platt. The laminae and overlying muscles were found to be infiltrated with material suggesting a new growth. Operation abandoned. Histological examination, however, confirmed the diagnosis of tuberculosis. Further operation not undertaken on account of widespread local disease.

Sept 22, 1933 —Patient died. At autopsy tuberculous lungs, mesenteric glands, and right kidney. Diagnosis of concertina collapse of Th 8 confirmed.

It is impossible to separate the effect on the cord of the lesion in the body of Th 8 from that due to the disease in the laminae and pedicles, but in view of the sudden deepening of the paralysis it is reasonable to suppose that the compression of the cord at the higher level was due to the rapid collapse of the body of Th 8 (Fig 541).

Acute Thrombosis of Vessels Supplying the Cord—The history in this condition is very similar and the prognosis is hopeless.

Case S 84—A boy, aged 18 years, admitted to St Luke's Hospital (London County Council), Lowestoft, under the care of Dr H J A Colvin, with caries of C 4-6. Enormous abscess in the left neck. Two months after he first came under observation complete paralysis of all four limbs suddenly developed, the condition resembling the 'spinal shock' of traumatic lesions. Death within three weeks; paralysis fixed to the last. At autopsy the large abscess was found to extend between the bodies of C 4 and 5 on to the anterior surface of the dura, but it is doubtful if it exerted any effective pressure on the cord, which was of normal contour; there was no obvious anatomical cause for the paraplegia. On opening the large abscess cavity to the left of the vertebral column, it was found that the wall of the abscess lay in intimate contact with the left vertebral artery, which contained a mural clot; the right artery was normal. Horizontal serial sections show that the clot had spread into the spinal branch of the artery that runs along the nerve-root and enters the cord at the level of the lesion (Fig 542); other smaller vessels around the nerve-root are thrombosed. Sections of the cord itself show that although the superficial vessels appear normal, there is widespread thrombosis of the vessels in the substance of the cord (Fig 543). The white matter is degenerate, though the grey matter, particularly in the anterior horns, appears normal; the central canal is patent. There is only slight dilatation of the vessels below the lesion.

The striking features of this condition—sudden complete paralysis, but nothing apart from the usual tuberculous lesion radiographically—are characteristic of a serious vascular lesion of the cord. Elsberg,⁸ though citing no evidence to prove his point, believes that this type of paraplegia is the result of a vascular lesion, and that operative interference is contra-indicated.

For the differentiation of these two serious accidents we are dependent upon X-ray examination. In concertina collapse the crushing of the vertebral body is easily identified, though it may be difficult to distinguish from the collapsed vertebra of secondary carcinoma. In acute thrombosis the tuberculous focus is of the usual type.



FIG 542—Case S 84 Infective thrombosis of the vertebral artery spreading along a spinal branch and involving most of the small vessels of the corresponding segment of the cord Horizontal section through the contents of the intervertebral foramen and the vertebral artery

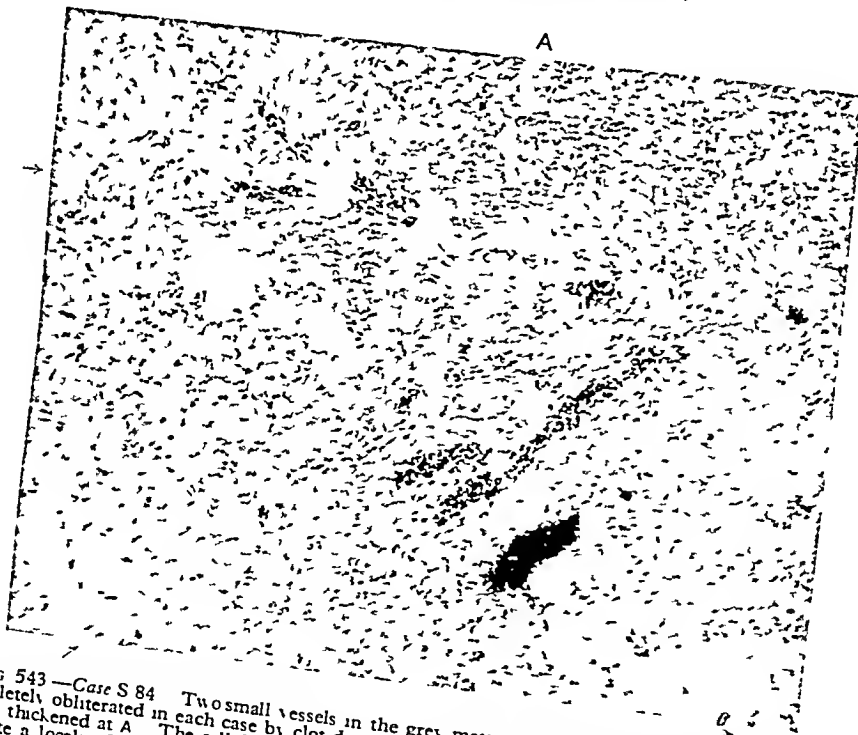


FIG 543—Case S 84 Two small vessels in the grey matter of the cord cut obliquely. The lumen is completely obliterated in each case by clot densely infiltrated with leucocytes. The wall of the upper vessel is thickened at A. The cellular infiltration of the area of grey matter shown is marked enough to constitute a localized myelitis. Most of the changes, however, were confined to the white matter

2 SPINAL TUMOUR SYNDROME

This puzzling condition (*Fig 544*), fortunately rare, is likely to be encountered by the neurologist rather than the orthopaedic surgeon, seeing that apart from operation it is indistinguishable from true spinal tumour. It is the only form of Pott's paraplegia in which neurological signs develop before radiographic changes are demonstrable in the spine. Case S 67 is a good example of this condition.



FIG 544 —From a woman aged 23 years, who died with paraplegia. Although there is widespread infection of the bodies of the 4th and 5th cervical vertebrae as evidenced by the loss of normal trabeculation in the spongy tissue, and some erosion of the posterior surfaces of both bodies, nothing abnormal could be found in the X-ray. The abscess responsible for the compression is well shown. (*Guy's Hospital Museum*)

Case S 67 —F G, a man 37 years of age, was admitted to the Westminster Hospital on June 7, 1926, under the care of Dr Hildred Carhill. There was a history of six months' backache, steadily becoming more severe, and increasing weakness in the lower limbs of two months' duration. On admission, paraplegia in extension with no voluntary power, cutaneous analgesia below Th 7. Cisternal CSF normal, lumbar CSF yellow, cells less than 1 per cmm, protein 0.35 per cent, globulin present, gold curve 000001223, WR negative. X-ray showed no bony lesion in spine, lipiodol completely arrested at level of Th 9, subarachnoid space displaced to right. Diagnosis, extra-medullary extra-theal tumour.

June 14, 1926 —Laminectomy by Mr Rock Carling. Th 9-12. Cord found displaced to right at level of Th 10, pulsation good above, poor below. During examination of theca, pus emerged from a mass of granulation tissue at the level of Th 12. Track found passing upwards for 1½ in on left of cord, i.e., to level of Th 10. Track irrigated through small catheter, wound closed. Material removed positive for TB. Paraplegia cleared up completely within eight weeks, but the laminectomy wound never healed and discharged large quantities of pus. Patient died in October, 1927, probably from amyloid disease. Before death, lesions involving the bodies of Th 7-8, 10-11 were demonstrated radiographically (at St Luke's Hospital, London County Council, Lowestoft).

In another case, S 80, a similar lesion was found in a man 21 years of age. However, in spite of early laminectomy, the paralysis was permanent, though the patient survived.

The spinal tumour type of Pott's paraplegia is a peculiarly lethal condition. The cases recorded by Fischer¹¹ and Vincent and Darquer³⁹ terminated fatally. I believe that Case S 67 is the only one described in which recovery from the paraplegia occurred before death, and this was undoubtedly attributable to the prompt operative intervention. The spinal tumour syndrome is therefore very definitely a Type II condition. recovery from paralysis has never been known to occur apart from operation.

Minimal Bone Destruction—It occasionally happens that paraplegia appears in cases where the amount of bone destroyed by the disease is extremely small. There were three such cases in my series. In Case S 14 the paraplegia was permanent; in Case S 5 the paraplegia took four years to clear up, then relapsed and persisted for a further six years; in Case S 7 (Fig 545) paraplegia was present for seven years. The association of slight bone destruction with permanent or long-standing paralysis seems to be more than a coincidence. Can it be that on account of the small amount of bone destroyed the vertebrae fall together prematurely and confine that part of the tuberculous focus that is the cause of the paraplegia to the spinal canal—in other words, reproducing the state of affairs found in the spinal tumour syndrome? It is probable that shrinkage of the contents of the intraspinal lesion is likely to occur more readily in a case where it communicates with the large fusiform or globular abscess usually found lying anterior and lateral to the vertebral bodies. The natural safety-valve for an intraspinal abscess is the channel that runs between the diseased bodies; if this is closed early, absorption of the contents of the focus is likely to be very slow.



FIG 545 Case S 7 Minimal bone destruction paraplegia for seven years

3 PARAPLEGIA IMPROVING, THEN RELAPSING AND REMAINING STATIONARY, OR MODERATELY SEVERE FROM THE FIRST AND SHOWING NO SIGNS OF IMPROVEMENT EVEN AFTER SEVERAL (4-7) MONTHS

Cases in this group belong essentially to Type I at first, and only too often gradually drift into Type II while the clinician is still fondly hoping that signs of recovery will ultimately appear. Every Type I paraplegia in which complete motor paralysis has been present for six months or rather less must be considered a possible Type II and should be investigated accordingly.

Good X-rays of the spine, including stereoscopic lateral views, should be taken and repeated if necessary at intervals of a few weeks. There are three possible explanations for the prolongation of severe paraplegia, and they will generally be revealed by the X-ray.

Pathological Dislocation (see Butler's article, p 753)—The presence of this

condition was proven in four of my cases and one of Butler's. A typical case has also been recorded by the Sorrels³¹ (p 412), although the pathology of the condition was not clearly recognized by them.

Case S 37—Complete motor paralysis in this patient with early active disease persisted unchanged for four months after she had been admitted to the Country Branch of the Royal National Orthopaedic Hospital. She was therefore well on the way to becoming a Type II paraplegia. Serial X-rays showed a fusiform thoracic abscess steadily increasing in size, and I suspected that this was the cause of the persistent paraplegia. On April 6, 1933 (four months after admission) I performed a costo-transversectomy, the abscess cavity contained only a small quantity of pus and caseous material—hardly enough to convince one that the abscess was really the cause of the trouble. One month later the paraplegia was quite unchanged. Re-examination of the lateral X-rays showed that there was probably a pathological dislocation, and stereoscopic lateral views taken on May 2 showed the dislocation clearly (*Fig 546*). This interpretation of the X-ray appearances was confirmed by Mr Bankart. The spinal canal was explored on May 4, 1933, and the cord was found tightly stretched over a sharp bony ridge.



FIG 546—*Case S 37*. Lateral X-ray of the spine of a patient with paraplegia resulting from pathological dislocation. The sharp bony ridge in the floor of the spinal canal is clearly visible at A. It was still more evident in the stereoscopic X-rays.

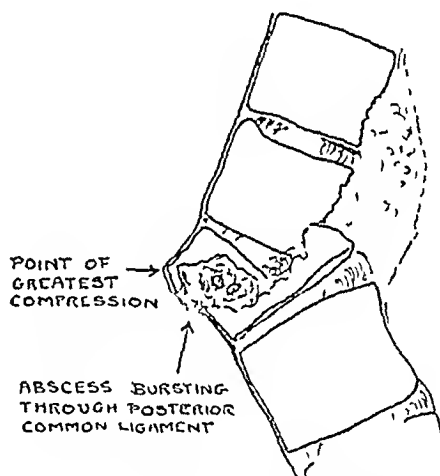


FIG 547—*Case S 37*. Diagram constructed from X-ray, operative, and post mortem findings.

If the existence of this condition is borne in mind, it should not be difficult to recognize it in the investigation of Type II paraplegias.

✓ **Compression by a Sequestrum.**—According to the Sorrels, the same severe persistent paraplegia may be caused by compression of the cord by a sequestrum. They have published an excellent example of this condition³¹ (*Figs 548, 549*), but they have not said how frequently a displaced sequestrum may be demonstrated radiographically. There are no cases of this type in my series, but a good one has been recorded by Butler (p 757).

In pathological dislocation and in compression by a sequestrum, the prognosis is always hopeless as regards the paralysis, and generally so as regards life.

✓ **Continued Activity of the Disease**—This is by far the most frequent cause

of persistence of an early paraplegia. There are certain cases in which the patient's resistance is so low that the disease in the spine progresses relentlessly in spite of the most efficient conservative treatment, and if paraplegia is present, it will continue in a severe form for an unusually long time, unless spontaneous drainage occurs as a result of external rupture or internal tracking of the abscess. Not infrequently there is complete motor paralysis for more than six months, and by the time activity of the disease finally abates, the cord is permanently damaged, and recovery from the paralysis will be incomplete or entirely absent.

Fortunately the signs of continued activity are very definite and one or more of them is present in every case. They are irregular fever, wasting, radiographic evidence of extension of the bony lesion and increase in size of the abscess shadow, and sometimes the appearance of fresh tuberculous foci in other parts of the body.

In my series alone there were 8 cases of continued activity of the disease which ended with permanent paralysis, and 6 in which the recovery was delayed and incomplete. A number of these cases ended fatally—for the reason that the disease progressed unchecked and finally became generalized.



FIG 548—The specimen from a case of the Sorrels (Brun—Pauline, aged 52 years). The sequestra lie in the cavity of an active abscess, but it is the bony fragments and not the abscess that are responsible for the compression. It will be noticed that the posterior arch of D 10 has slipped down behind the arch of D 11, the spinal canal is therefore narrowed to some extent, a contributory factor in the compression of the cord.

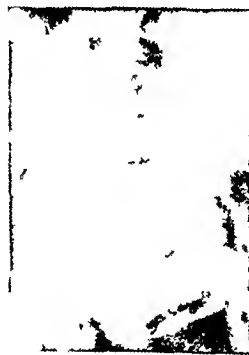


FIG 549—An X-ray of the case shown in Fig 548 taken during life. The large sequestrum is shown very clearly. (Figs 548, 549 are reproduced by kind permission of Professor E. Sorrel and Masson et Cie, Paris.)

In posterior spinal disease with paraplegia, the condition may follow a Type I or Type II course, depending on the virulence of the infection. In one of my cases, S 36 (Fig 540), the paraplegia was never complete and lasted for only eight months in all. In the other, S 64, the resistance of the patient was poor from the beginning, and paraplegia developed early in the course of the disease. Voluntary control was soon lost, and never returned until a few months before the patient died of phthisis and amyloid disease, twenty-one months after he first came under observation.

TYPE III PARAPLEGIA

Type III paraplegia is immediately differentiated from Types I and II by the history and clinical picture.

The story is generally as follows—early in life the patient has suffered with spinal caries, as a result either of extensive disease or inadequate treatment, or both, bone destruction has been excessive, and a number of vertebral bodies, generally from four to nine, have been completely destroyed. The disease finally healed and the patient led a normal life, nothing unusual was noticed for a number of years apart from slowly increasing deformity of the spine, often so marked as to attract the attention of the patient and his relatives. Then after a long period, anything from four to forty years, the patient complains of a little weakness of the lower limbs or inco-ordination when walking. This marks the onset of the paraplegia. Pain is rarely felt, but if it is present, it takes the form of backache or irregular pains radiating down the lower limbs, only one patient in my series suffered with girdle pains.

On examination a severe deformity is found, local tenderness is generally absent—in fact there is nothing apart from the paraplegia to suggest active disease. The paraplegia is usually incomplete, but may be severe.

Butler has shown that in this large group of cases the majority are suffering from a recrudescence of disease in the neighbourhood of the spinal cord, in a smaller number there is evidence that points to stretching of the cord over a bony ridge in the floor of the spinal canal—a paraplegia of purely mechanical origin. It is most difficult, often impossible, to distinguish these two varieties of late onset paraplegia, but a consideration of the following points is sometimes of value—

1. There may be clinical and radiographic evidence of active disease—further erosion of the vertebral bodies, the shadow of a recent abscess, or, as in one of my cases, S 23, a fresh abscess discharging on the surface.

2. The absence of a bony ridge in the floor of the spinal canal clearly excludes bony compression, but the converse is not always true—the presence of a ridge does not necessarily indicate mechanical compression. In one of my cases, S 22, a bony ridge was demonstrated radiographically and seen at operation, but the real cause of the paraplegia was renewed activity of the disease.

3. If the patient is treated by a method that tends to decrease the angulation of the spine (e.g., suspension on a Fisher frame), it would be reasonable to suppose that a paraplegia due to mechanical compression would either improve or, at least, remain stationary. If, in spite of such treatment, the paraplegia become steadily worse, the cause must be infective. In a recent case, S 110, a late onset paraplegia steadily increased in spite of suspension at operation the spinal theca was found embedded in recent tuberculous granulation tissue.

The prognosis as regards life in late onset paraplegia is good. Even patients who become permanently paralysed rarely die as a result of the paralysis. The deformity of the spine and chest is probably a greater menace to life. Carey Coombs⁶ pointed out the association of sudden fatal cardiac failure with severe kyphosis, but this is quite apart from the incidence of paraplegia.

The recovery rate in Butler's and my series of Type III paraplegias is as follows—

	UNDER 16 YEARS				OVER 16 YEARS			
	R	W	B	HJS Total	R	W	B	HJS Total
Type IIIa (recovered from paralysis)	24	+	11	= 35	10	+	12	= 22
Type IIIb (paralysis permanent)	6	+	6	= 12	13	+	7	= 20
Recovery rate	74.5 per cent				52.4 per cent			

Any patient under 16 years of age with late onset paraplegia has, therefore, an excellent chance of recovery—about 75 per cent. In later years the prospect is not so good—about 50 per cent.

We are unable to differentiate the prognosis in late onset paraplegia due to renewed activity of the disease from that due to compression by bone, seeing that in most of our cases it was impossible to be certain which of the two causes was operative.

Nor are we able to state in advance what chance of recovery there is likely to be in any particular case. The only reliable guidance is that given by the neurological features of the paraplegia (p 770). Any case in which complete motor paralysis has been present for more than six months and the vibration sense lost is unlikely to recover completely. Butler has drawn attention to the composition of the cerebrospinal fluid in late onset paraplegia, and the possible association between a high protein content and a favourable prognosis. This observation may prove to be of value, but we have not yet examined a sufficient number of cases to justify any dogmatic statement.

SUMMARY OF PROGNOSIS

A guide to prognosis is given based on the neurological features of Pott's paraplegia, irrespective of the cause. It may be epitomized in the following way:

VERY FAVOURABLE	UNFAVOURABLE	HOPELESS
Paraplegia in extension	Sphincter involvement	Paraplegia in flexion
Voluntary control never lost	Urinary infection	Flaccid paralysis (except in cauda equina and brachial lesions)
Voluntary control absent for not longer than 6 months	Pregnancy	Loss of voluntary control for longer than six months
Absence of sensory loss		Loss of vibration sense
Absence of sphincter involvement		Old age

All these signs are of value in determining when a Type I paraplegia is changing to Type II, though they do not indicate the reason for the change. Similarly, they show when a Type IIIa case is likely to develop into a Type IIIb.

A clinical approach to the differentiation of Type I from Type II paraplegia is described. The histories of the various Type II paraplegias are highly characteristic.

A complete paraplegia of rapid onset is typical of concertina collapse of a vertebral body, or thrombosis of vessels supplying the cord. The two conditions are distinguished radiographically.

The spinal tumour syndrome is clinically unlike any other form of Pott's paraplegia, the only condition with which it may be confused is true spinal tumour.

A Type I case continuing into Type II—that is, remaining paralysed completely for six months or longer—is due either to pathological dislocation of the spine, or to compression of the cord by a sequestrum, or to continued activity of the disease. Again, these three conditions are differentiated radiographically.

It may be objected that as several of the Type II conditions are so uncommon, too much attention has been paid to them in this paper, and that the suggestions that have been made are unlikely to be of real value in clinical practice. We must, however, bear in mind that these conditions, rare singly, when taken together

account for 21 per cent of all paraplegias occurring during the active stage of the disease, and they are responsible for many tragedies. Admittedly the outlook for the patient with Pott's paraplegia is good, but it might be better, and, as will be shown in the next section of this paper, a rational plan for the prevention of permanent paralysis can be based only on a careful analysis of the atypical cases, among which the Type II paraplegias form an important group.

It is possible that further investigation will establish other causes of Type II paraplegia. Several conditions described in the literature have been excluded by us on the grounds of insufficient evidence. We have confined our attention entirely to conditions in which the underlying pathology has been definitely established.

As regards Type III paraplegia, we are still very much in the dark both in our methods for differentiating the "renewed activity of disease" type from the less common type with bony compression of the cord, and also in giving a reliable prognosis.

A few signs are of value, but they are present in only a minority of the cases.

Part II PREVENTION AND TREATMENT

TYPE I PREVENTION

Seeing that paraplegia is not infrequently the first clinical sign of spinal caries, the prevention of Type I paraplegia is not always possible. This is particularly the case in adults, in whom the disease often continues for many months with relatively little bone destruction, no deformity, and few symptoms. However, early symptoms in the form of local or referred pain are generally present, and too often overlooked by the clinician. Spinal caries as a cause of backache in adults is insufficiently recognized. It seems as if *deformity* stands in the foreground of the commonly accepted clinical picture of the disease. Actually, in adults, obvious deformity is rare and pain almost invariable. In two of my cases, both adults, upper abdominal pain led to diagnoses of gall-stones in one case, and duodenal ulcer in the other. The first was treated by cholecystectomy, the second by dieting. It was not until paraplegia developed that the real cause of the pain was discovered.

In children, on the other hand, paraplegia as a first sign is rare. The deformity due to bone destruction develops early and rarely escapes recognition, and backache—uncommon in children—immediately suggests a serious spinal lesion.

Nor in the established case of spinal caries is the prevention of Type I paraplegia always possible. By pure ill-luck, the primary focus may lie far back in a vertebral body, or in a lamina, and spread rapidly into the spinal canal. In other cases the virulence of the infection may be such that the patient never has the slightest chance of recovery: the disease spreads rapidly, the cord is inevitably involved, and the patient soon succumbs to the generalized disease.

As far as we know, there is only one special measure applicable during the course of ordinary conservative treatment that is of definite value in diminishing the incidence of paraplegia—namely, hyperextension of the spine.

It is certain that extensive destruction, and probable that the formation of large abscesses, occur more readily in any case of joint tuberculosis in which movement, particularly friction between joint surfaces, is allowed during the stage of activity. In hip disease we know that bone destruction and abscess formation are reduced

to a minimum by combined traction and fixation. The same is true of spinal caries. In the spine firm fixation is not difficult to maintain, but it is impossible

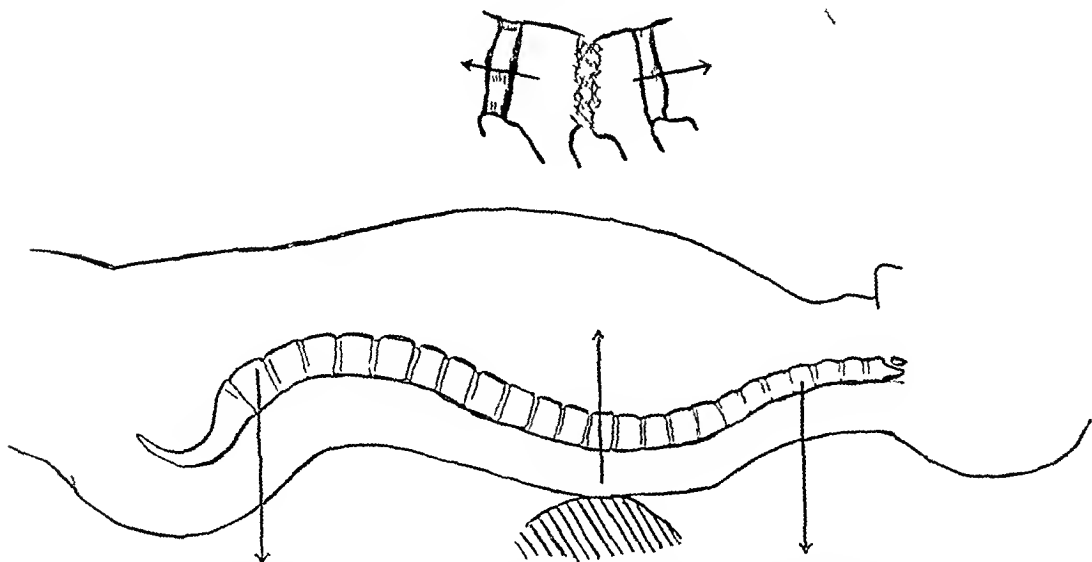


FIG 550—The simplest form of hyperextension of the spine. Pressure is made at the site of the lesion, with the patient lying supine. The extending force is the body weight acting above and below the lesion. There must always be a pressure point at the level of the lesion. If this is absent, as in the hyperextended anterior shell, hyperextension results only in the development of compensatory curves.

to apply efficient longitudinal traction to the thoracic spine where bone destruction is often so marked. However, the same effect may be obtained without difficulty by hyperextension, provided that constant pressure is applied at the level of the lesion (Fig 550). When the thoracic spine in a case of active disease is hyperextended, the involved vertebrae tend to separate, not infrequently radiographically visible separation actually occurs. In any case the vertebrae are relieved from longitudinal pressure, and the grinding resulting from the constant respiratory movements is reduced. I have been able to observe the amount of bone destroyed in a large number of cases of thoracic caries in children, some of whom were treated in plaster beds, and others in hyperextension. In the latter the bone loss was considerably less than in the first group. Hyperextension is only necessary for as long as there is radiographic evidence of active

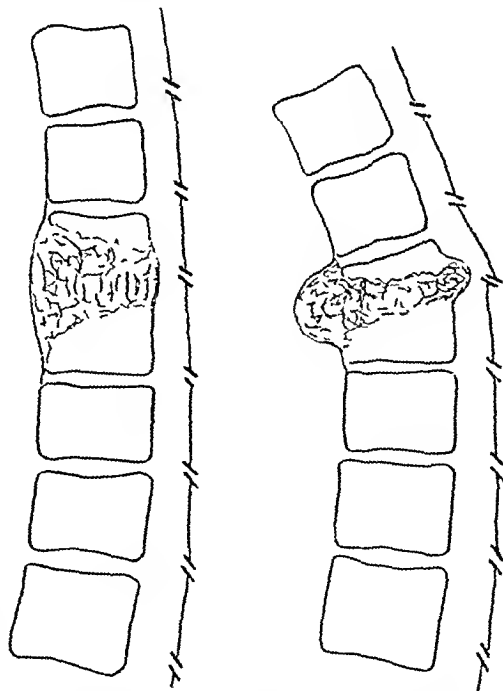


FIG 551—It is possible that when inflexion of the spine occurs in an active case of caries, the contents of the abscess are squeezed circumferentially and in encroaching on the spinal canal may compress the cord. Probably a reversal of this process accounted for the rapid recovery in a number of Goldthwait's cases of paraplegia that were treated by gentle hyperextension of the spine under anesthesia.

spreading disease. As soon as activity has abated, hyperextension should be discontinued and the diseased vertebræ allowed to fall together. Healing will then occur with the loss of fewer vertebral bodies than would have been the case under treatment in simple recumbency. Furthermore, it is probable that the abscess so constantly present is favourably influenced by hyperextension both as regards size and shape. Goldthwait¹¹ found that if a patient with active disease and paraplegia was anæsthetized, extension of the spine could easily be obtained by gentle pressure on the kyphos, and not infrequently the paralysis disappeared, often with remarkable rapidity. He was unable to offer any explanation for this phenomenon. It can only be explained by the influence of the extension of the spine on the shape of the abscess (*Fig 551*), and if this suggestion is correct, it furnishes another powerful argument in favour of hyperextension of the spine in the prevention of Type I paraplegia.

TYPE I TREATMENT

The treatment of almost every form of Pott's paraplegia is primarily conservative.

Fixation—If treatment in hyperextension is called for in uncomplicated thoracic disease, it is all the more necessary if paraplegia is present. Any apparatus complying with the conditions already described (*see Fig 550*) may be used. In my experience the Bradford frame is most satisfactory. Adults who cannot tolerate frame treatment may find a hinged plaster bed more comfortable. Heavily padded frames of the Robert Jones pattern, while providing excellent fixation, are not to be recommended in cases of thoracic disease, on account of the difficulty of ensuring that the spine is adequately hyperextended.

Unfortunately, the paraplegia is sometimes so severe that treatment in hyperextension cannot be continued on account of the rigidity of the lower limbs and the tendency to trophic sores. Such cases must be nursed in an ordinary plaster bed, or, if very severe, in a reversible spinal bed. In mild cases the lower limbs may be left free. The moment spasticity becomes marked, adhesive extension must be applied to the lower limbs and long metal splints to hold the feet at right angles and the knees in extension. It is advisable to have the splints bent to an angle of 10° or 15° at the knees, full extension is uncomfortable and may lead ultimately to the development of back-knee. Fixation of the lower limbs must be maintained until spasticity has almost disappeared. In the most severe cases extensions and splints are not tolerated. The spasm is uncontrollable, and if efforts to keep the lower limbs straight are continued, severe trophic sores are certain to develop. In these cases the lower limbs must be left free, and every joint put through its full range of movement twice a day in order to prevent the development of contractures.

Acute retention requires prompt catheterization under rigidly aseptic conditions. Continued retention is uncommon, and I have never found it necessary to catheterize a patient repeatedly, or evacuate the bladder by manual expression as recommended by the Sorrels. In all cases with bladder involvement, it is wise to administer some form of urinary antiseptic from the first, whether infection is present or not. Severe constipation should be controlled by enemata.

Massage has no place in the treatment of established paralysis unless there-

is a lower motor neuron lesion. In the ordinary type of spastic paraplegia, physical treatment is called for only when the paralysis is well on the way to recovery, at this stage massage and exercises are of great value in getting rid of the last traces of inco-ordination and clonus. In lower motor neuron lesions, where muscular wasting is present from the first, the treatment is the same as in a case of infantile paralysis during the stage of recovery.

Operation.—Although spontaneous recovery is assured in almost every Type I paraplegia, there are certain cases bordering on the Type II "continued activity of disease" group, in which the paralysis is so profound and prolonged that the patient is reduced to a condition of extreme misery and the surgeon to a state of constant anxiety. In these cases operation is of value.

It has frequently been observed that if an abscess appears superficially in a case of active disease with paraplegia, the latter begins to clear up rapidly as soon as the contents of the abscess have been evacuated. There is an undoubted connection between drainage of the focus of disease and relief of the paralysis. Several useful procedures which may be classed together as lateral drainage operations have been evolved from this observation.

LATERAL DRAINAGE OF A SPINAL ABSCESS. COSTO-TRANSVERSECTOMY.—Although others before Menard²⁰ had performed this operation, he was the first to demonstrate its value in Pott's paraplegia. By 1900, when he published his famous book, *Etude pratique sur le Mal de Pott*, he had performed costo-transversectomy 23 times. In 13 cases recovery commenced immediately after the operation, in 4 after one week, and in 2 at the end of three months. There were 5 failures, 2 due to compression by bone, and 3 in which the cause of failure was unknown.

Operative Technique.—According to Menard, the operation should be performed on the left side. A transverse incision, 5 to 7 cm. in length, is made over the vertebral end of the rib corresponding to the apex of the kyphos. After the rib has been exposed, the periosteum is carefully elevated on the superficial and deep surfaces. The rib is divided with nibbling forceps at a point about 4 cm. from the tip of the transverse process of the corresponding vertebra. The transverse process is then cut through at its base and removed after its costal attachment has been divided. The inner end of the rib is now lying in a periosteal tube attached only by its head. It is removed by combined twisting and traction. Sometimes the removal of the rib opens the abscess cavity. If it does not, the tunnel should be gently explored with the finger, and when the wall of the abscess is located it is incised with a blunt instrument.

Menard does not mention ligation and division of the neurovascular bundle in a case where more than one rib has been resected. I have found that a most satisfactory exposure is obtained if the neurovascular bundle is encircled with a double ligature, tied in two places, and divided.

When it is proposed to resect more than one rib a longitudinal incision in the line of the costo-transverse joints may be employed with advantage.

ASPIRATION OF A THORACIC ABSCESS.—This form of treatment was introduced as an alternative to costo-transversectomy, the great virtue claimed for it being that the risk of secondary infection is almost entirely eliminated.

In 1908 Finck¹⁰ reported the successful aspiration of a mediastinal abscess.

Schede²⁷ describes a most exact method for determining the depth of an abscess. The necessary calculation is distinctly complicated, but he claims that

if the trocar and cannula are introduced in the way that he describes, nothing but the abscess will be encountered

Calve³ believes that inflexion of the vertebral column at the site of a thoracic lesion squeezes an abscess laterally and posteriorly. It is, therefore, a mistake to hope to relieve tension in the part of the abscess that is compressing the cord by introducing a trocar and cannula along the side and to the front of the vertebral bodies. He introduces the instrument through the intervertebral foramen into the abscess as it lies directly anterior to the cord. Though the method may be sound in theory, most of us would say with Delbet, "Je n'ai pas cherché à en évacuer par la technique de Calve qui me partait au-dessus de mes forces"

The most attractive method is that described by Valtancoli³⁷. He takes antero-posterior and lateral X-rays of the spine in the usual way, and then with barium in the œsophagus. Having localized the abscess exactly, he introduces a slightly curved trocar and cannula. The instrument must not be less than 9 cm in length, and the bore of the cannula not less than 3 mm. The instrument is passed close to the upper border of the rib in order to avoid the intercostal vessels, then towards the mid-line, aiming directly at the vertebral body. Pus is usually found at a depth of from 6 to 8 cm.

The disadvantages of mediastinal aspiration are obvious. Puncture of the lung and even the aorta (!) have been reported, but no one confesses to any serious trouble having followed such accidents.

It is a matter of history that Menard finally abandoned costo-transversectomy on account of the fatalities that resulted from infection of the sinus that he deliberately produced by operation. However, those who have followed him have not all had the same unfortunate experience. Jackson Clarke⁵ (1903), Wassiliew⁴⁰ (1909), Steindler³⁴ (1929), and Girdlestone¹³ (1931) have expressed themselves in favour of the operation and have not considered secondary infection a serious menace. Indeed, it never occurred in most of the cases that these observers have recorded. No doubt this was due in part to the practice (of Girdlestone, for example) of suturing the wound after evacuation of the abscess, but the real secret of their success was that adequate conservative treatment was continued for many months after the drainage operation had been performed. As Girdlestone so properly insists, costo-transversectomy is only an incident in the treatment. Most of Menard's patients were allowed to walk the moment the paraplegia had cleared up, and in the light of our present knowledge it is quite evident that they were never given a real chance to recover from the active disease in the spine.

We are of the opinion that costo-transversectomy is of very definite value in certain cases of paraplegia of early onset. It is probably the best of all the operations that have been proposed and practised *

* *Laminectomy*—Before 1900, Menard had reached the conclusion that laminectomy was of very limited value in the treatment of Pott's paraplegia. His views have been upheld so repeatedly in papers written since that time, that a detailed discussion of the operation is no longer necessary. Unfortunately, in spite of the mass of adverse evidence, laminectomy for severe Pott's paraplegia still finds a place in the teaching and practice of many surgeons. As will be shown later, the operation may be of some value in certain rare forms of paraplegia, but as a routine procedure it is to be condemned. See Table II, p. 796.

Spinal Fusion—De Courcy Wheeler⁴¹ and others are of the opinion that patients with paraplegia recover more rapidly after a fusion operation has been performed. The evidence

The advantages claimed for costo-transversectomy are —

1 It attacks the main cause of the paraplegia—the abscess arising from the focus of disease in the spine

2 Emptying of the abscess cavity reduces the pressure on the cord and may also reduce the toxicity of the focus—which, as Butler has shown, is probably of considerable importance in determining changes of conductivity in the cord

3 After costo-transversectomy, drainage of tuberculous material is away from the cord—not around it, as after laminectomy

4 The operation does not weaken the bony spine, as does laminectomy

5 The technique is not particularly difficult, nor is there any great operative risk

6 A relatively large drainage canal is created, through which it is possible to evacuate not only fluid pus, but granulation tissue, bone sand, and caseous material. This cannot be accomplished by any of the methods of aspiration that have been described

7 Any further collection of pus that may form after costo-transversectomy will come to the surface at the site of operation. If the wound has been sutured, aspiration may safely and easily be performed at a point just to one side of the incision

If a sinus forms, it should be regarded as beneficial rather than dangerous. It indicates that continuous drainage is needed for a longer or shorter period, and the sinus will close as soon as the lesion in the spine begins to heal. Secondary infection did not occur in any of our cases.

Drainage of a cervical abscess causing pressure signs is exactly analogous to costo-transversectomy, and need not be described separately.

It has already been shown that owing to continued activity of the disease, a certain number of cases that commence as Type I end as Type II, compression and intoxication of the cord continue for so long that irreparable damage is inflicted. It is therefore necessary to consider together the operative treatment of Type I and the treatment of Type II due to continued activity of the disease.

Costo-transversectomy is indicated in profound Type I paraplegia. Though recovery may possibly be expected to occur spontaneously, there is no reason why the patient should be allowed to suffer for many months if a simple and safe operation can give him relief.

Case S 24 —A girl, 8 years of age, was admitted to the Country Branch of the Royal National Orthopaedic Hospital in July, 1929, with spinal caries of about four months' duration. The X-ray showed an early lesion of Th 9-10 with a small fusiform abscess.

November, 1929 —X-ray, abscess larger

October, 1930 —Onset of paraplegia

During the following eleven months the paraplegia became more and more profound until the limbs were held rigidly in extension, voluntary power was lost, and sensation (except vibration) absent below the level of Th 10. Serial X-rays showed a steady increase in size of the abscess. It was decided that costo-transversectomy should be performed, but the parents obstinately refused to give their consent.

supporting this view is not convincing, and in America, where fusion operations are often performed early in the course of the disease, many cases have been found (by Steindler and Silver, for example) to develop severe paraplegia after the performance of a fusion operation. Two cases in my series, Nos S 45 and S 75, developed paraplegia after 'successful' fusion operations had been done. The value of the fusion operation in cases with paraplegia has not yet been proved.

February, 1932—Patient removed from hospital against advice, and nursed at home During the following two years she was visited regularly

August—A large abscess appeared below the left 12th rib

October—Abscess ruptured, discharging at least a pint of pus Almost immediately the paraplegia began to clear up

September, 1934—Good voluntary power, but still a little spasticity

In this case, fortunately for the patient, spontaneous drainage occurred, but it is clear that had operation been performed in 1931, she would have been spared twelve months' misery Furthermore, it is probable that the paralysis would have cleared up completely after operation, whereas now the lower limbs are still a little spastic The following case provides a happy contrast

Case S 38—A girl, 12 years of age, was admitted to the Royal National Orthopaedic Hospital (Great Portland Street) in September, 1922, under the care of Mr Jackson Clarke, with two months' history of trouble in the back and weakness of the lower limbs On admission she was found to have an early lesion of Th 6-8, and well-marked but not severe paraplegia

November, 1922—Complete loss of voluntary power, anaesthesia below Th 8, and incontinence of urine

Dec 4—Costo-transversectomy (Mr Jackson Clarke) Large abscess opened

Dec 19—Complete sensory recovery, a little voluntary control of the toes No incontinence

Dec 27—Wound soundly healed Transferred to Country Branch, Stanmore, conservative treatment continued

During the following two years, the abscess reappeared and was aspirated repeatedly The paraplegia cleared up completely

September, 1925—Discharged, walking well Condition since this date most satisfactory

This is an example of a perfectly managed case As soon as it became evident that the paraplegia was certain to be severe and prolonged, operation was performed As a result the patient was completely paralysed for only two months The costo-transversectomy ensured that further collections of pus would make their way to the surface and not towards the spinal canal they were dealt with successfully by aspiration Conservative treatment was continued for an adequate length of time after operation

Clearly, then, we are not justified in temporizing with any severe Type I paraplegia, for it may develop into a hopeless Type II case Operation should not be delayed for longer than six months after voluntary control has been completely lost,* provided that the X-ray shows a definite abscess shadow It has already been shown that spontaneous recovery after more than six months of complete motor paralysis will be either incomplete or entirely absent

Several of my cases were operated on too late—eighteen months, two years, or even three years after the onset of complete motor paralysis—in no case with the slightest benefit (see Table I, p 795)

A further indication for operation is evidence of pressure on other vital structures—with or without paraplegia In one of our cases, a large cervico-mediastinal abscess was the cause of severe paraplegia and marked respiratory obstruction Complete relief rapidly followed incision and evacuation of the abscess

* It must be emphasized that paraplegia of only six months' duration is not necessarily an indication for operation Paraplegia may be present for as long as two years, and yet voluntary control may never have been completely absent

TYPE II PREVENTION

From all that has been said of the pathology of concertina collapse of a single vertebral body, pathological dislocation, and compression of the cord by a sequestrum, it is clear that these conditions can generally be prevented by prolonged hyperextension of the spine. This is in fact the only non-operative measure of any value in the prevention of Type II paraplegia, but unfortunately the condition of the patient is sometimes so critical that this ideal method of nursing cannot be employed—and only too often the accident that damages the cord beyond hope of recovery has occurred before the patient comes under treatment.

TYPE II TREATMENT

In thrombosis of vessels supplying the cord and in concertina collapse of a vertebral body, no radical treatment is of any avail. Early laminectomy is indicated in all cases of spinal tumour syndrome, though it has yet to be shown that the condition may be completely cured by surgical intervention. Costo-transversectomy is indicated in all cases with continued activity of the disease, and there is abundant evidence to show that recovery will occur, provided that the

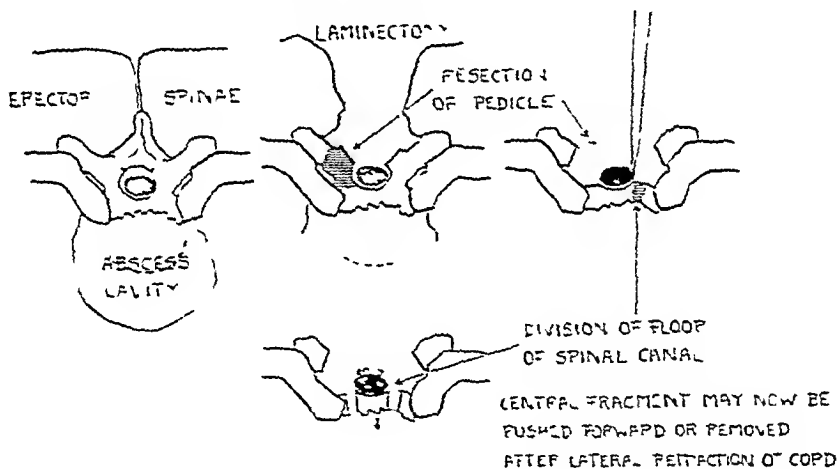


FIG 552.—In pathological dislocation the cord is stretched over a sharp ridge in the floor of the spinal canal. The ridge is formed by the remains of a vertebral body which is part of a more or less complete bony ring encircling the spinal cord. In order to decompress the cord the ring must be broken anteriorly. The last step in the operation is not shown—division of the one intervertebral disc that holds the offending fragment to its neighbouring vertebral body. This should present no technical difficulties.

operation is performed early and that the patient does not succumb to generalized tuberculosis. The details of treatment have already been discussed.

No case of compression by sequestrum has yet been dealt with successfully by operation. Theoretically, it would seem possible to remove even a large sequestrum from the front of the cord by a large costo-transversectomy approach. In Case S 58 a sequestrum was thought to be the cause of a severe persistent paraplegia: a dense irregular shadow was visible in stereoscopic lateral X-rays lying immediately anterior to the cord in the centre of an extensive tuberculous focus. After resection of two ribs and ligation of the intervening neurovascular bundle, a good exposure of the focus was obtained. The 'sequestrum' proved to be a large dense mass of caseous material, which I removed without difficulty. If

the mass had been a sequestrum, it could probably have been removed just as easily, and I see no justification for the pessimism of the Sorrels about this condition.³¹ The primary difficulty is in recognizing the presence of a sequestrum at an early date.

In pathological dislocation radical operative treatment may hold out some prospect of relief, and although I have no records of successful operative intervention, it must be evident that an operation based on a clear understanding of the morbid anatomy of the condition is at least worth a trial.

As the fragment of bone responsible for compression of the cord is part of a vertebral body, it must at the same time form part of a bony ring encircling the

spinal canal. The integrity of this ring will depend upon the extent to which the pedicles have been eroded by the disease. If the anterior half of the ring could be resected, the cord would be liberated, and this should be the aim of any operation designed for the relief of this condition. The steps of such an operation are shown in *Fig 552*. In *Case S 37* (see *Fig 546*) I performed a laminectomy and found the cord stretched over a bony ridge in the floor of the spinal canal; the right pedicle attached to the ridge was resected and an attempt was made to remove the latter. This step appeared to be successful, and the theca, which up till that moment had been white, tense, and pulseless, became pink, lax, and pulsatile. Unfortunately, there was no improvement in the paraplegia after operation, and the patient died of acute tuberculous pneumonia and sepsis from sores seven weeks later. At the autopsy it was found that the bony ridge had been incompletely removed (*Fig 553*), the left half was still present and the cord stretched over it. It is probable that the angulation increased after the operation, and that an incomplete removal of the ridge, apparently adequate at the time of operation, did not afford permanent decompression of the cord. It is on this failure that the suggested operation is based. Due consideration has been given to the possibility of a postero-lateral attack on the ridge by an approach resembling a costo-transversectomy, but



FIG. 553—*Case S 37*. The cord and the remains of the bony ridge, which is compressing the cord on its left side.

as the ridge may be attached to both of its pedicles, it must be clear that the only reasonably safe and sure method of removal of the triangular fragment is in the way suggested.

Among German surgeons—Tillmanns,³⁶ Borchers,² and Nureddin,²³ for example—there is remarkable unanimity in advising laminectomy for disease commencing in the neural arches—posterior spinal disease. Operation is indicated even in the absence of paraplegia. It is the surgeon's one opportunity for performing a complete excision of the diseased bone in a tuberculous spine. If a radical

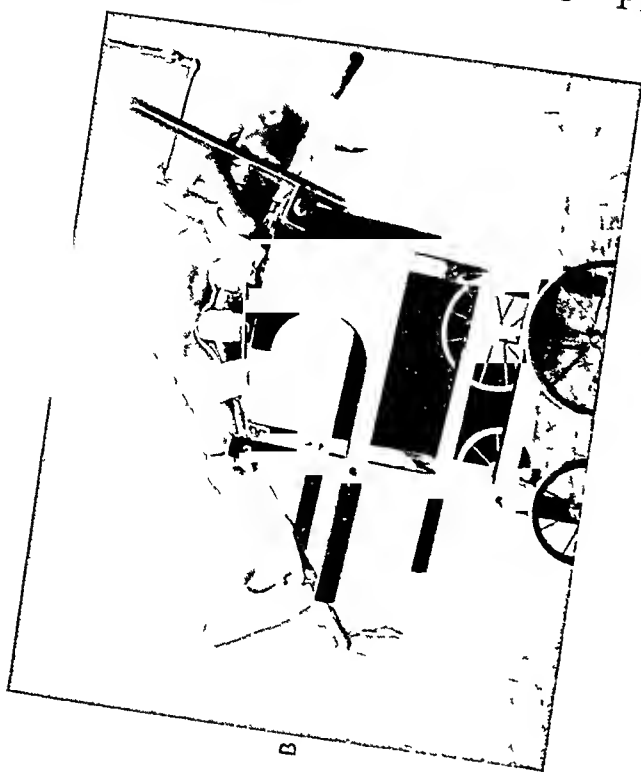
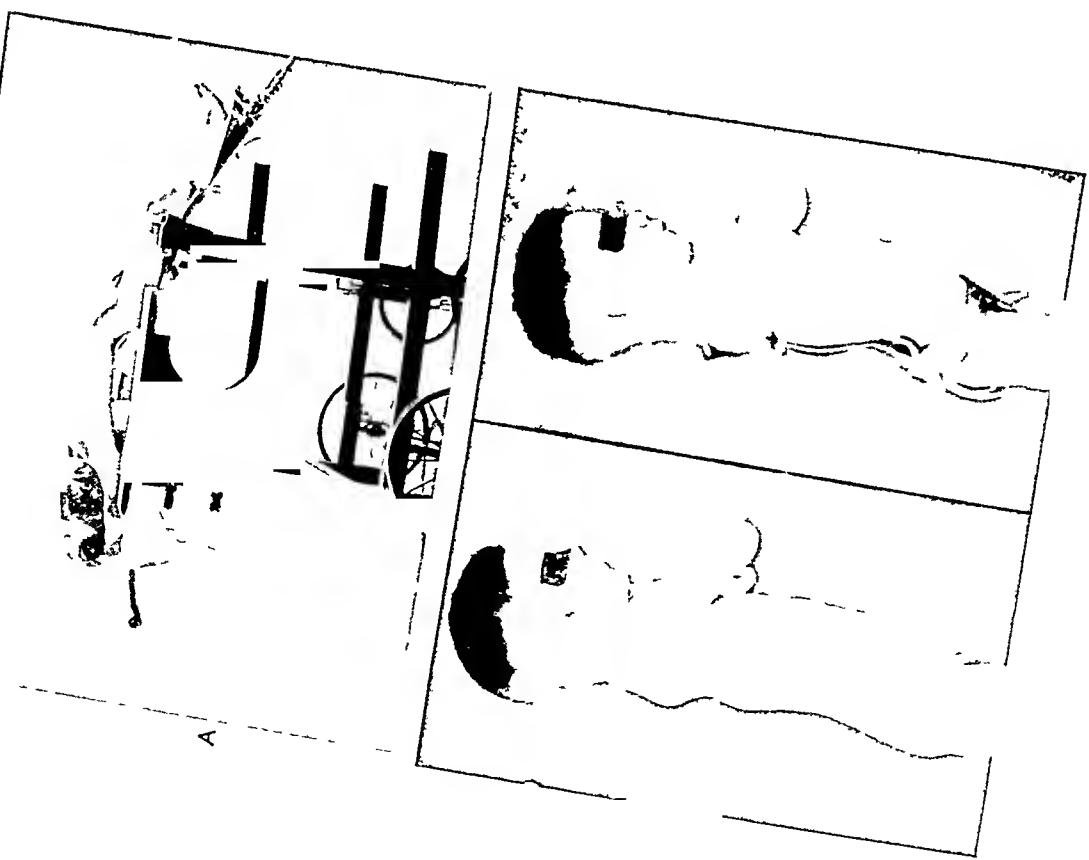


Fig 554—An example of the production of a well-balanced spine as the end result of treatment of extensive low thoracic disease in a child A T R, aged 4, at onset of disease No compensatory curves B Two years later The kyphos now falls into the hollow between the two cross pads of sponge-rubber and there are excellent compensatory curves above and below C After three years in recumbency (two and a half years with the cross padding) the general balance of the spine is excellent He is seen standing with and without a Jones' back brace (Patient under the care of Mr R W Butler, to whom I am indebted for the photographs and X-ray—Fig 555)



operation is not performed, infection of the bodies will almost invariably follow, as in two of my cases. Successful operations have been recorded by Sharpe²⁹ and Pels-Leusden,²⁵ though in the case reported by the latter a lesion appeared later in the bodies of Th 12 and L 1, in spite of a complete excision of the initial focus in the arch of L 1.

TYPE III PREVENTION

Late onset paraplegia occurs only after severe and extensive disease of the thoracic spine which has led to the development of considerable deformity. The



FIG 555 —X ray of spine of patient shown in Fig 554

prevention of this very common variety of paraplegia is dependent upon the efficiency of the treatment of the active disease. The frequency of late onset paraplegia (accounting for about 50 per cent of all cases) is a reproach on the management of spinal caries in this country.

The essentials of efficient treatment are —

- 1 Reduction of bone destruction to a minimum. This is best accomplished by nursing the patient in some form of hyperextension apparatus.
- 2 Prolonged treatment in recumbency until long after bone destruction has

ceased and signs of repair are apparent radiographically. Again it must be emphasized that in most cases the cause of late onset paraplegia is renewed activity of the disease. In a number of our cases it was very evident that the disease had never really healed; the patients were not kept on their backs for a sufficient length of time. In some cases four years' continuous treatment is essential.

3 When bone destruction has come to an end, simple hyperextension—which holds the focus open—should be discontinued and the involved vertebræ allowed to fall together and fuse. At the same time compensatory curves must be induced above and below the lesion, so that the patient will have a balanced spine when he gets up (*Figs 554, 555*). The nearer the centre of gravity approaches to the spinal column, the less leverage will there be at the site of the healing lesion, and the more the compressive force tending to bring the involved vertebræ firmly together, so encouraging fusion.

4 At this stage some form of fusion operation may be performed to strengthen the weak point, but before this is done the involved vertebræ must be in good contact with each other, and compensatory curves must have been produced.

Late onset paraplegia rarely occurs after efficient treatment of the active and healing tuberculous spine.

TYPE III TREATMENT

The treatment of late onset paraplegia is essentially conservative. Prolonged rest in recumbency at an open-air hospital is necessary, as there is usually a recrudescence of active disease in the spine. Hyperextension is of very doubtful value, and there are no rational grounds for employing it.

In a certain number of cases (25 per cent in children, 50 per cent in adults) it becomes evident, as treatment is continued, that recovery is unlikely to take place with purely conservative measures. The question of operative intervention must be considered. We must remember that these cases are bad risks—whatever operation, spinal or abdominal, is performed on them. There is gross deformity of the chest, the vital capacity is diminished, and there is generally some degree of cardiac inefficiency. As late onset paraplegia is rarely fatal of itself, operation should therefore be advised only with great caution.

If a fresh paravertebral abscess is clearly demonstrable, costo-transversectomy is indicated (*Table I, Case B 45*—p 795), though generally the reactivated focus is confined to the spinal canal.

Laminectomy is usually the only hope, but there is a danger peculiar to this operation. Examination of many specimens from cases of late onset paraplegia shows that the integrity of the spinous processes and laminae (which are often solidly fused) is sometimes the only factor preventing gross dislocation of a severely damaged spine. If laminectomy is performed (without subsequent grafting), the bony spine is virtually transected and subluxation may occur, and so lead to even more extensive damage to the cord than was present before operation. In some of our cases this actually happened, either soon after operation or when the patient was allowed to sit up in a support, and the last state of that man is worse than the first. Quite apart from this risk, the removal of epidural granulation tissue or simple decompression of the cord for late onset paraplegia is not often followed by recovery. Operation was successful in only 2 out of 9 of our cases. Radical treatment may sometimes be justifiable, but only when requested by the patient or his

relatives after the poor chances of success have been fully explained. The operation itself should always be combined with grafting.

It is possible that operation will prove more successful in cases where the cord is stretched over a bony ridge. Simple laminectomy is futile (*see Fig 538 and Table II, Cases S 43 and S 100—p 797*). Nothing less than the removal of the hump over which the cord is stretched will suffice.

In *Case S 82* there was good reason to suppose that bony compression was the cause of a late onset paraplegia (*Fig 556*). The patient was at first under my care,



FIG 556—*Case S 82*. A woman of 42 years. Apparently healed thoraco-lumbar caries, paraplegia of gradual onset occurring about thirty years after the disease had been active. Lateral X-ray taken after injection of lipiodol via cisterna magna, shows incomplete thecal block—probably due to projection backwards of bony spur at A.

but was transferred later to Mount Gold Hospital, Plymouth, under the care of Mr Norman Capener. I suggested to him that if the paraplegia did not clear up under conservative treatment, operation might be worth considering. Some months later he wrote to say that as the woman's condition was not improving, he had explored the spinal canal. His account of the operation is as follows —

Curved incision to right of kyphos. Skin-flap turned medially. At apex of kyphos right sacrospinalis divided transversely and retracted upwards and downwards. Laminae, transverse processes of three or four vertebrae stripped subperiosteally on right side only. The two central transverse processes were then excised together with the lateral portions of the laminae almost to the roots of the spinous processes. Corresponding pedicles resected. The intervening nerve-trunk, which appeared to be the first lumbar, was divided about $\frac{3}{4}$ in from the theca and used as a means of rotating the theca backwards. On account of the deformity, the bodies of the vertebrae came well into view and the region of the posterior and right lateral surfaces was readily explored. Absolutely no evidence of active tuberculous disease was found. At the apex of the deformity the intervertebral spaces could be recognized but were apparently completely ossified. There seemed no doubt that the lesion was

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Table I—Results of Costo-transversectomy

Case No.	Type	Age	Duration of Paraplegia before Operation	Operative Findings	Result		Comments
					Life	Paraplegia	
B 42	I	17	7 months	Large abscess much pus and caseous material	Recovered	Recovered	Sinus formed closed later
B 44	I	47	5 months	Much pus and caseous material	Recovered	Recovered	
B 46	I	38	4 months	Much pus and caseous material	Recovered	Recovered	
B 48	I	26	3 months	No abscess very active tuberculous granulation tissue	Recovered	Permanent	
S 38	II	12	3 months	Large abscess	Recovered	Recovered	Operation should not have been performed no radiographic evidence of abscess
S 58	II	14	6 months	Much caseous material and bone sand	Recovered	Recovered	Many aspirations afterwards, continued activity of the disease
S 46	II	3	6 months	Large cervico-mediastinal abscess bulging into pharynx	Recovered	Recovered	Sinus formed and discharged for many months after operation Continued activity of the disease
S 49	I	16	2 months	Large cervical abscess	Recovered	Recovered	Severe dyspnea in addition to paraplegia Cervical abscess opened later Sinus formed and closed disease
B 45	III ₁	37	4 months	Much pus and caseous material	Recovered	Recovered	Evacuation of abscess through cervical incision
B 47	II	28	4 months	Large abscess	Recovered	Recovery then relapse	Sinus formed closed later
S 31	II	11	13 months	Thick walled abscess containing solid caseous material	Recovered	Permanent	Laminectomy later—failure second costo-transversectomy a definite case of continued activity of the disease
S 41	II	10	2 years	Thick caseous material and bone sand	Survived	Permanent	Operation delayed too long Laminectomy was performed after the eight days after operation Death
S 102	II	12	2 years	Much caseous material	Survived	Permanent	case of continued activity of the disease
S 37	II	15	7 months	Very little caseous material and pus	Died tuberculous pneumonia	Permanent	Operation delayed too long Continued activity of the disease
						Permanent	Operation delayed too long Continued activity of the disease
						Permanent	Pathological dislocation cause of paraplegia Costo-transversectomy should never have been performed

Table II—RESULTS OF LAMINECTOMY

CASE No	TYPE	AGE	DURATION OF PARAPLEGIA BEFORE OPERATION	OPERATIVE FINDINGS	RESULT		COMMENTS
					Life	Paraplegia	
B 49	I	46	About 8 months	Prominent mass of tuberculous debris and bone anterior to cord, dura very vascular and covered with tuberculous granulation tissue	Recovered	Recovered	Inadequate treatment before operation, laminectomy performed just as good conservative treatment was commenced. Recovery would probably have occurred apart from operation
S 31	I	11	1 year 2 months	Cord compressed against laminae by abscess	Post-operative death	>	
B 50	II	40	4 months	No pulsation of cord opposite Th 8	Died	Permanent	
S 14	II	23	3 years 4 months				Minimal bone destruction (<i>see p 777</i>), operation should have been performed earlier
S 34	II	30	1 year 5 months	Apparently healthy pulsating theca pushed back to some extent, further exploration of spinal canal abandoned on account of patient's poor condition	Died	Permanent	Continued activity of disease
S 44	II	9	6 months	Cord pushed back, and partially surrounded by granulation tissue	Survived	Temporary recovery then permanent	Continued activity of disease
S 66	II	29	1 month		Died	Permanent	Very active disease
B 56	II	25	3 years	Cord tightly stretched over a large angulation	Survived	Permanent	Pathological dislocation
S 37	II	15	7 months	Cord tightly stretched over angulation removal of bony ridge attempted	Died	Permanent	Pathological dislocation
S 62	II	16	3 months	Abscess surrounding cord which was stretched over a bony ridge in	Died	Permanent	Pathological dislocation

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		16	1 month	Granulation tissue in spinal canal cord stretched over angulation	Died	Permanent	Pathological dislocation
S 67	II	37	2 months	Granulation tissue and abscess	Died	Recovered	Spinal tumour syndrome
S 80	II	21	1 month	Granulation tissue	Survived	Permanent	Spinal tumour syndrome
S 61	IIIa	37	2 months	Abscess anterior to cord	Recovered	Recovered	Postero-lateral approach laminar
S 22	IIIa	10	6 months	Granulation tissue around cord	Recovered	Recovered	Postero-lateral approach laminar
S 82	IIIa	42	8 months	Bony ridge in floor of spinal canal removed	Recovered	Recovered	Postero-lateral approach laminar
B 51	IIIb	27	6 months	Granulation tissue and caseous material removed from epidural space pulsation returned to cord	Survived	Slight improvement	Postero-lateral approach laminar
B 54	IIIb	35	1 year	Epidural space full of dense granu- lation tissue	Survived	Slight improvement	
B 55	IIIb	44	4 to 5 years	No evidence of pressure by bone and no active tuberculous material found	Survived	Slight improvement	
B 57	IIIb	27	3 months	Cord buried in caseous material and pus, most of which was removed pulsation in cord did not return	Survived	Permanent, worse after operation	Subluxation of spine when patient got up
B 58	IIIb		2 years	Abscess pushing cord backwards contents removed	Survived	Improved, then permanent	
S 32	IIIb	26	2 years	Granulation tissue	Survived	Permanent	
S 43	IIIb	12	5 months	Cord stretched over bony ridge	Operative death		
S 100	IIIb	14	1 year 10 months	Cord stretched over large bony ridge	Died	Permanent	
S 110	IIIb	9	8 months	Granulation tissue	Survived	Worse	

quite healed After the cord had been rotated and the extradural stripping of the posterior surface of the bodies carried inwards anterior to the cord, a definite bony spur was encountered During the stripping of this spur sharp spasms of the legs were produced The spur appeared to be projecting from the posterior inferior edge of a vertebral body slightly cephalic to the actual apex of the kyphos This spur was cut away with a chisel, the muscles were drawn together, and the wound closed

The night following the operation the patient had no spasms for the first time for many months Her improvement was very obvious and has continued ever since

SUMMARY OF PREVENTION AND TREATMENT

	PREVENTION	TREATMENT
<i>Type I—</i>	Not always possible Hyperextension of definite value	<i>a</i> Primarily conservative <i>b</i> Costo-transversectomy in any severe case likely to become Type II 'continued activity of disease' <i>c</i> Incision of abscess in cervical cases, particularly if there is also marked pharyngeal obstruction
<i>Type II—</i>		
Acute thrombosis of vessels supplying the cord	Not possible	Operation contra-indicated
Concertina collapse	Hyperextension	Operation contra-indicated
Spinal tumour syndrome	Not possible	Laminectomy
Continued activity of disease	Costo-transversectomy while still Type I	Costo-transversectomy
Compression by sequestrum	Hyperextension	Removal of sequestrum should be attempted
Pathological dislocation	Hyperextension	Removal of bony ridge possibly of value
Posterior spinal disease	Not possible	Laminectomy
<i>Type III—</i>	Efficient treatment of the tuberculous spine at every stage	Conservative In severe cases, possibly laminectomy and graft removal of tuberculous material or of bony ridge if mechanical compression is present

It has been shown that the efficient treatment of uncomplicated spinal caries is of value in preventing the onset of paraplegia—particularly Type III

When paraplegia is present the treatment is primarily conservative In severe Type I cases timely costo-transversectomy is of value, not only in shortening the duration of paralysis, but in preventing the development of Type II paraplegia due to continued activity of the disease

The less common forms of Type II paraplegia, although very grave conditions, may possibly yield to operative treatment

In Type III paraplegia we are almost confined to conservative treatment, so far operation has yielded but few successes

Laminectomy as a routine operation for severe paraplegia is to be condemned, the results are appalling However, there are a few uncommon but very definite indications for its performance

The writers of the two preceding papers desire to express their thanks to the many surgeons who gave permission for their cases of Pott's paraplegia to be included in the present series, also to their assistants, and the members of the administrative, nursing, and laboratory staffs whose help has been so valuable

While it is impossible to mention all by name, they wish to thank particularly the staffs of the Royal National Orthopædic Hospital, Queen Mary's Hospital, Carshalton, St Luke's Hospital, Lowestoft, Princess Mary's Hospital, Margate, The Royal Sea Bathing Hospital, Margate, The Alexandra Hospital for Hip Disease, Swanley, St Vincent's Orthopædic Hospital, Pinner, The Shropshire Orthopædic Hospital, Oswestry, and the Hôpital Franco-Américain, Berck-sur-Mer. Their thanks are also due to Dr J G Greenfield, Professor H A Harris, and to the curators of the museums of the London Medical Schools for permission to investigate museum specimens and for generous assistance in studying material from their own cases.

REFERENCES

- ¹ ANDRE-THOMAS, *Rev neurol*, 1924, II, Feb, 342
- ² BORCHERS, E, *Munch med Woch*, 1924, lxxi, 652
- ³ CALVE, J, *Rev neurol*, 1923, xxxix, 711
- ⁴ CHARCOT, J-M, *Leçons sur les Mals du Systeme nerveux*, 1880, II, 88 Paris
- ⁵ CLARKE, J JACKSON, *Practitioner*, 1903, lxxi, 407
- ⁶ COOMBS, CAREY, *Brit Jour Surg*, 1930, xviii, 326
- ⁷ DOLLINGER, B, *Die Behandlung der tuberculosen Wirbelentzündung*, 1898, Stuttgart
- ⁸ ELSBERG, C A, *Tumours of the Spinal Cord*, 1925, New York
- ⁹ FICKLER, A, *Deut Zeits f Nervenheilk*, 1900, Ivi, 1
- ¹⁰ FINCK, E, *Beitr z klin Chir*, 1908, lxx, 65
- ¹¹ FISCHER, O, *Med Klink*, 1919, xv, 1284
- ¹² FORESTIER, J, *Le Tron de Conjugaison vertebra et l'Espace epidural* 1922 Paris
- ¹³ GIRDLESTONE, G R, *Brit Jour Surg*, 1931, xix, 121
- ¹⁴ GOLDTHWAIT, J E, *Trans Amer Orthop Assoc*, 1899, xli, 91
- ¹⁵ GOWERS, W, *Diseases of the Nervous System*, 1892, I London
- ¹⁶ GREENFIELD, J G, and CARMICHAEL, E A, *The Cerebrospinal Fluid in Clinical Diagnosis*, 1929 London
- ¹⁷ HASSIM, G B, *Histopathology of the Peripheral and Central Nervous Systems*, 1933 London and New York
- ¹⁸ KAHLER, O, *Zeits f Heilkunde*, 1882, III, 187
- ¹⁹ LANNELONGUE, *Tuberculose vertebrales*, 1888, Paris
- ²⁰ MENARD, V, *Etude pratique sur le Mal de Pott*, 1900 Paris
- ²¹ MICHAUD, "Sur la Meningite et la Myelite dans le Mal vertebra", *These de Paris*, 1871
- ²² MYERS, T H, *Trans Amer Orthop Assoc*, 1891, IV, 124
- ²³ NUREDDIN, A, *Deut Zeits f Chir*, 1926, cxcviii, 125
- ²⁴ PAINTER, C F, and MOORE, G C, *Amer Jour Orthop Surg*, 1910, viii, 306
- ²⁵ PELS-LEUSDEN, *Deut med Woch*, 1918, xlv, 143
- ²⁶ POTT, P, *Remarks on that Kind of Palsy of the Lower Limbs which is Frequently Found to Accompany a Curvature of the Spine, and is Supposed to be Caused by it Together with its Method of Cure* Tract, London, 1779
- ²⁷ SCHEDE, F, *Munch med Woch*, 1922, lxxix, 779
- ²⁸ SCHMAUS, H, *Die Compressions-myelitis bei Karies der Wirbelsaule*, 1890 Wiesbaden
- ²⁹ SHARPE, N, *Amer Jour Surg*, 1923, xxxvii, 142
- ³⁰ SORREL-DEJERINE, *Contribution a l'Etude des Paraplegies pottiques*, 1925 Paris
- ³¹ SORREL, E, and MME SORREL-DEJERINE *Tuberculose osseuse et osteo-articulaire*, 1932 Paris
- ³² SORREL, E, and MME SORREL-DEJERINE *Rev neurol*, 1924, July, I
- ³³ SOUQUES, A, *Ibid*, 1923, lxxix, 649
- ³⁴ STEINDLER, A, *Diseases and Deformities of the Spine and Thorax*, 1929 London
- ³⁵ STRUMPELL, A, *Lehrbuch der speciellen Pathologie und Therapie*, 1890, II, Chap III
- ³⁶ TILLMANS, H, *Arch f klin Chir*, 1903, lxxv, 134
- ³⁷ VALTANCOLI, S, *Ibid*, I and 244
- ³⁸ VACCHELLI, S, *Ibid*, I and 244
- ³⁹ VINCENT, C, and DARQUIER, *Rev neurol*, 1925, xxxii, 100
- ⁴⁰ WASSILIEW, M A, *Arch f klin Chir*, 1909, lxxxviii, 845
- ⁴¹ WHEELER, W I de C, *Robert Jones Birthday Volume*, 1928 London
- ⁴² ZEIGLER, *Lehrbuch d pathol Anatom*, 1885, II, 627

SKELETAL LIPOID GRANULOMATOSIS

(Hand-Schüller-Christian's Disease)

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INTRODUCTION

It has long been recognized that certain disturbances of lipid metabolism may constitute a pathology which creates important considerations of diagnosis and treatment, and, while there is an underlying factor common to all the conditions grouped under this heading, there are certain distinctions which justify the adoption of an individual specification. The distinctions are based upon a variety of factors—differences in the biochemical agencies which appear to initiate disease, in the type of tissue affected and the reaction which these tissues display, in the prognosis, and in the response to therapeutic measures. It is upon such grading that a subdivision has been based which includes at present some five varieties of disturbance of lipid metabolism. They are *Gaucher's syndrome*, in which a lipo-protein of the cerebroside type initiates a morbid anatomy, *Niemann-Pick's syndrome*, associated with a phosphatid lipid tissue deposit, *Tay-Sachs' syndrome*, probably attributable to a cerebroside protein derangement, *Hand-Schüller-Christian's syndrome*, in which an error in cholesterol metabolism is apparently the factor responsible, and a somewhat *heterogeneous group*, the distinctions of which have not yet been fully investigated, but which may be said to include xanthoma, xanthomyeloma, and xanthelasma, conditions which are attributable to degrees of cholesterolaemia. If an attempt is made to summarize the factors which exert a common and universal influence in what seems to be a concatenation of diseases, it will be found that for certain obscure reasons a state of affairs arises in which an excess of lipid circulates in the body fluids. A proportion of the excess is taken up by body cells of the histiocyte type, and thereafter a series of cell changes constitutes a pathology which is reflected in various clinical signs, in questions of diagnosis, of prognosis, and of treatment.

The purpose of this paper is to present a study of certain aspects of Hand-Schüller-Christian's syndrome, more particularly in respect of the changes which affect the skeleton, and in so far as it is a lipid derangement associated with the formation of granulation tissue, and because our interests are more particularly in relation to bone changes, it seems appropriate to use the title of *skeletal lipid granulomatosis*.

It may seem at first sight that the surgical interests of the subject are by no means evident, but reflection will show that the matter is one of real surgical importance. The criticism may be offered that the condition is so rare that it scarcely justifies a detailed or serious consideration, and the fact that no detailed publication on the subject exists in British literature would seem to support this

view, but the probability is that the belief in its rarity has been exaggerated, for, if a single surgical charge encounters three cases within as many years, the likelihood is that the occurrence of the disease is being overlooked, that the diagnosis is being confused, and that consequently treatment is not always appropriate. For these reasons alone the subject is worthy of consideration, and, if further justification is needed, it may be found in the fact that here is a subject of immense pathological interest, a matter which may well have far-reaching association with problems of tissue reaction and tumour growth.

CASE REPORTS

I feel that it will stress the practical significance of the problem to recount a summary of the clinical histories of four cases which have been under observation in the past few years.

Case 1—In June, 1928, it was noticed that a female, aged $1\frac{9}{12}$ years, of unexceptional personal and family history, had developed a limp in relation to the left hip. Examination appeared to exclude the possibility of a primary joint lesion, but radiography revealed that the left iliac bone was the site of an extensive rarefactive process. There was no doubt that this lesion, whatever its nature, was the factor responsible for the limp, and, as the diagnosis was uncertain, a biopsy was carried out. Histological investigation revealed the picture of a lipoid granulomatosis showing the characteristic lipoid-containing histiocytes and multinuclear giant cells, features which will be fully discussed when the histopathology is described. I confess, however, that at this stage the histological diagnosis was not established, and it was only in the light of subsequent experience that the real character and significance of the picture were appreciated. In the beginning the appearances were mistaken for those of myeloma, and the questions of prognosis and treatment were oriented accordingly. The further history was that bone changes of a similar nature appeared in relation to the right ilium, the right femur, the bodies of the vertebrae, and the calvaria (Fig 557).

Treatment was instituted by radium packs applied over the affected bones, and by deep radiotherapy, the view being taken that the case was one of multiple tumour formation of the myeloma type. Under this treatment the child gradually improved, recalcification appeared in the rarified zones, and to-day she is in good health, although the areas originally affected still show extensive lacunization. In this instance the error in diagnosis entailed the omission of special lines of investigation, and the case record is therefore imperfect, but, reviewing the condition in the light of fuller knowledge, there appears to be no doubt that the condition was one of lipoid granulomatosis of the cholesterol type.

Case 2—A male, aged 3 years, was admitted to hospital in November, 1932. It was stated that the first sign of health disorder was noted at the age of 2 years, when the teeth became affected by what was thought to be an extensive infective process. The teeth were extraordinarily soft, crumbling down to the level of the gums, and in several instances were extruded from what appeared to be pockets of inspissated pus. The stature and bony



FIG 557—*Case 1* Radiogram illustrating defects in the left parieto-occipital region

development were below normal, and the child was described as being irritable and unhappy. These features indicative of bad health were attributed to dental sepsis, though it seemed difficult to understand why such persistent and widespread tooth infection should exist in a child of such tender years. In October, 1932, it was observed that the child was crying a great deal, and from time to time placed his hand over the left ear, as though experiencing pain in that region.

On examination there was found a fluctuating swelling immediately above the left mastoid, and, on the supposition that this was an abscess, the swelling was explored by means of an aspirating needle. A small quantity of faintly blood-stained fluid was removed, and on examination showed the characteristics of cerebrospinal fluid. At this point the child was brought under our observation. He was found to be under weight (38 lb) and poorly developed, and those who were concerned with the care of him in hospital were soon made aware that he was unusually fractious and irritable. The faeces showed slight undue

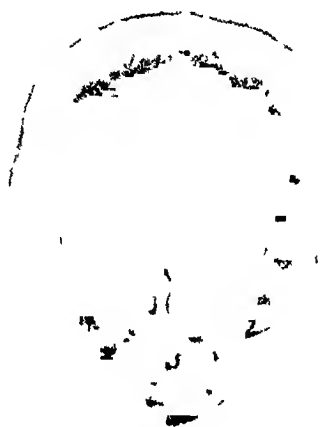


FIG 558—Case 2. Antero-posterior radiogram of skull showing defects in left temporal region and left half of the mandible.



FIG 559—Case 2. Lateral radiogram of skull showing extensive calvarial defects in occipital and left temporo-sphenoidal region.

projection of both eyes. On examining the skull there was found on the left side an extensive osseous defect involving the greater wing of the sphenoid, the squamous portion of the temporal, and the occipital bone. In this area the pulsation of the underlying brain was palpable and visible. The left half of the mandible was enlarged, as though it were the site of a central cyst or tumour formation. A routine examination of the various systems was carried out, it yielded a negative result except in so far as the urinary system was concerned, in relation to which diabetes insipidus was revealed, 75 oz. of urine being passed in twenty-four hours, with a specific gravity of 1004. The entire skeleton was radiographed, and there was found an extensive lacuna in the left speno-temporo-occipital region with smaller lacunæ in the frontal, the right parietal, and the occipital regions (Figs 558, 559). The swelling of the left half of the mandible was shown to be due to a central cyst expanding the cortex.

I confess we were at a loss to know the explanation of the various findings, or to trace the thread by which they might be linked up and associated, and it was at this stage that we were fortunate enough to enlist the interest of Mr. David M. Greig, the Curator of the Royal College of Surgeons Museum in Edinburgh. He taught us wisdom on many points, and it was he who suggested that the case under observation might be an example of the Schuller-Christian syndrome, or, as he preferred to designate it, skeletal lipoid granulomatosis.

Further investigation was accordingly made. The cell picture of the blood was normal, the blood calcium was slightly raised (11.6 mgrm. per cent), the calcium output of the urine

was normal, the blood-cholesterol was raised to 267 mgrm per cent. One of the skull lacunæ was explored, there was found to be complete disappearance of the ossified tissue, the gap being occupied by a quantity of yellowish rubber-like material which was closely adherent to the underlying dura mater. This tissue on being submitted to microscopical examination showed the characteristic appearance of a granulomatous disturbance, while the presence of histiocytes, foam cells, and multinucleated giant cells made it clear that the lesion was of the Schüller-Christian type.

The fate of this child was extremely unfortunate. After a course of deep radiotherapy, from which he derived considerable benefit, he returned home, to succumb to an attack of post-influenzal pneumonia in February, 1933.

Case 3—A male, aged 4 years, born in India, of whose early medical history no more need be said than that he was described as an underdeveloped and somewhat irritable baby. He had the reputation of being a remarkably thirsty little creature, and it may be allowed that he deserved such a description when I say that he sometimes consumed three quarts of water in twenty-four hours. In December, 1932, a swelling the size of a cherry was discovered over the centre of the occipital bone. It was believed to be a sebaceous cyst, but, as it was increasing in size, its removal was decided upon, and this was carried out in January, 1933. Operation revealed that the diagnosis was erroneous, the tumour was a solid one, and not only so but it was associated with disappearance of the underlying bone, while it showed evidence of involving the dura mater. The appearance suggested a malignant condition, and, when the tissue was submitted to microscopical examination, the worst fears appeared to be confirmed, for the pathologist reported that the tumour was a sarcoma. About this time what seemed to be irrefutable confirmation of the malignant character of the lesion was demonstrated when a tumour of the left clavicle, assumed to be a secondary deposit, made its appearance.

In view of the gravity of the prognosis the parents were advised to return to this country as soon as possible, and the child came under our care in March, 1933. The appearances by that time were in many respects characteristic. The child was nervous and irritable, he was small for his age and considerably under weight (his weight at 4 years being 22 lb and his height 2 ft 8½ in), he was particularly resentful of any attempt at examination, his eyes showed a slight degree of exophthalmos, the scalp veins were unduly prominent, particularly when the child strained or cried. On handling the skull there was found to be an extensive defect of the calvaria in the occipital region, the area measuring 4 cm by 5 cm, there was a smaller lacuna in the centre of the left parietal bone. The left half of the mandible was enlarged, while the medial half of the left clavicle showed a fusiform enlargement the size of a bantam's egg. No abnormality could be detected in the rest of the osseous system. The teeth were extremely unhealthy, many being worn to the gum level, while the two anterior to the molar teeth in the left half of the mandible were obviously loose, and it was noticed that when they were 'rocked' by the finger a small quantity of yellowish inspissated material resembling pus appeared around the edges. The posterior chain of the deep cervical lymph nodes on both sides of the neck was enlarged, each gland being about the size of a hazel nut, there was no pain associated with the enlargement, and there was no evidence of periadenitis. The respiratory, circulatory, and alimentary systems appeared to be normal. Investigation of the urinary system yielded some interesting results. There was a pronounced polyuria, an average of 90 oz being passed in twenty-four hours, and in relation to this degree of polyuria there was a corresponding degree of polydipsia, over 100 oz of fluid being consumed in the same period of time. The blood showed a normal picture so far as red and white cell counts and hæmoglobin readings were concerned, but a biochemical investigation revealed a definite increase in the lipid content, the readings being as follows: Total cholesterol 236 mgrm per cent, total fatty acid 287 mgrm per cent, lecithin 274 mgrm per cent. This result revealed a moderate degree of cholesterolaemia if one accepts the normal cholesterol content of the blood as 100 to 150 mgrm per cent.

A radiographic investigation of the entire skeleton was carried out. The occipital and right parietal bones were the sites of extensive lacunæ, the edges of which showed a characteristic irregular map-like outline (*Fig 560*), the centre of the body of the left half of the mandible was occupied by a cyst-like formation, and it was interesting to notice that the apices of certain of the teeth appeared to lie within the confines of this cavity. The medial half of the left clavicle contained a central cyst-like deposit, which was causing expansion

of the cortical bone and absorption of the cancellous interior. The remainder of the osseous system appeared to be healthy.

At this stage an opportunity was afforded of examining sections of the skull tumour which had been removed in India in January, 1933, and it was found that the microscopical picture was characteristic of a lipoid granulomatosis of the Schüller-Christian type.

The diagnosis of the condition appeared to be fully established, and under a scheme of treatment which included deep radiotherapy and pituitrin injections the child improved. He is at the present date in reasonably good health, but in the progress there have been two episodes which are significant of the disease and of the way in which it may be associated with problems in local diagnosis.

In October, 1933, pain was complained of in the left half of the mandible, and on the supposition that there was a tooth infection, the left canine of the first dentition was removed. It was found that the buried portion of the tooth was covered with a yellow caseous-like substance, while a quantity of a similar material (which as a matter of fact was mistaken for pus) escaped from a cavity in the jaw with which the apices of the tooth had been communicating. A subsequent analysis of this substance showed that it was largely composed of cholesterol. (See Fig 565.)

FIG 560—Case 3. Lateral radiogram of skull showing extensive calvarial defects.

In November, 1933, the child developed the signs and symptoms of acute right mastoid infection. Operation was carried out, when there was found to be an extensive infiltration of the mastoid sinus and the squamous portion of the temporal bone by a deposit similar to that which had been encountered in relation to the teeth. The appearances suggested inspissated pus, and no doubt there was a certain element of infection, but analysis showed that the deposit was largely composed of cholesterol.

Case 4—I am permitted to quote this case by the courtesy of Dr. Leila Hawksley, Pathologist to the Cancer Hospital, Fulham Road, London, and Professor Woodburn Morrison. I take the opportunity of expressing my indebtedness to them for allowing me to study the case history, and examine various radiograms and pathological material illustrative of the case.

A female, aged 8 months, was brought to the Out-patient Department of the hospital in January, 1932, with the history that a month previously it was noticed that she was not using the right arm freely. Examination showed that there was a swelling involving the infraspinous area of the right scapula (Fig 561), it was ovate, some 60 mm in length, and on radiography it was found to be associated with an expansion and erosion of the bone in an area immediately inferior to the glenoid cavity. No other bones were affected, the various systems appeared to be healthy, there were no blood changes, no polyuria, and no disturbance of the eyes.

FIG 561—Case 4. Radiogram showing the tumour of the right scapula. (By permission of Professor Woodburn Morrison.)

On Jan 14 the swelling was explored, and a portion removed for microscopical examination. The histopathology illustrated appearances suggestive of a lipoid granulomatosis, that is to say, lipoid-containing cells of the histiocyte type and multinuclear giant cells. The tumour was treated by radium applied interstitially, a dosage of 2,568 radium-hours being given.

In July, 1932, the child was re-examined. The swelling of the right scapula persisted, and there was found a small tumour arising at the centre of the left parietal bone, while the calvaria in an area immediately underlying the tumour had disappeared. The child was kept under observation, and by November, 1932, further bone deposits appeared in the skull, the left scapula, the right ilium (Fig 562), and the first rib the skull deposits being associated with palpable areas of tumour formation.

Meantime the child's general health showed deterioration, but there was no evidence of any visceral or blood change, the Wassermann reaction was negative. Further bone deposits now made their appearance in the skull (Fig 563), the left mandible, the left clavicle, and the right ilium. There is no record that polyuria existed.



FIG 562—Case 4. Illustrating the defect in the deposit in the neck of the (By permission of Professor Woodburn Morrison)

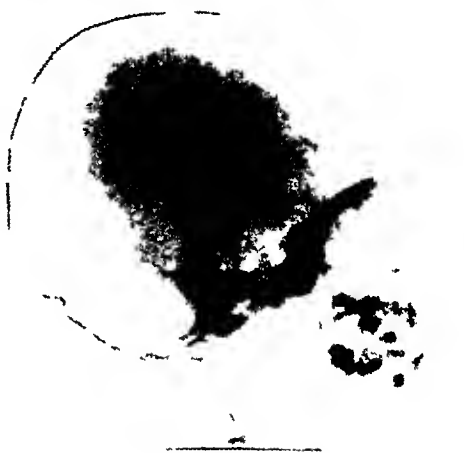


FIG 563—Case 4. Radiogram of the skull. The chart-like outline of the skull defects is well shown (By permission of Professor Woodburn Morrison)

Deep radiotherapy was begun in November, 1932, and from that time improvement became evident. The last report indicated that the skull tumours had disappeared, and that the lacunæ were being replaced by new bone. The other bone tumours also showed signs of improvement, and the child appeared to be in good general health and developing normally.

These are the presentations of four cases which show certain features in common—deposits, chiefly in the membrane bones, of such a nature as to be mistaken for malignant tumour formation, and illustrating a pathology which, while it bears resemblance to primary tumour formation, appears to have its explanation in a stimulus of reticulo-endothelial tissues by a chemical substance of a lipoid character. The cases may be regarded as illustrating the disease in the early stages of its progress, and, as we shall see, a later and more extensive pathology implies a wider and more varied clinical history.

THE HISTORICAL ASPECT

Before discussing the various aspects of the condition which these cases illustrate, I propose to present a short historical account of the disease, for it is interesting to recall the phases and stages by which one's knowledge of the disturbance has advanced

Priority of recognition is generally ascribed to Alfred Hand,¹ who published in 1893 a case showing a *trilogy of features*—calvarial osseous defects, exophthalmos, and polyuria. The condition was undoubtedly one of lipoid granulomatosis, but Hand, misled by the caseous-like appearance of the granulation deposit, attributed the changes to tuberculosis. Literature, however, contains an earlier record, and, though the story is incomplete and the diagnosis was unsuspected, there is abundant interest in the facts. I am indebted to Mr David M Greig for bringing this case to my notice. The details are as follows —

In 1865 Thomas Smith,² of London, examined a child 4½ years old who had developed a swelling over the occipital bone. The swelling was described as soft

and fluctuant, and on the supposition that it was an abscess, Smith was asked to operate upon it. He found, however, that, though fluctuant, it also pulsated, while there was an evident deficiency in the underlying skull. He therefore refused to incise the swelling. Other areas of deficiency appeared in the calvaria, and, when some months later the child succumbed to an attack of whooping-cough and Smith had an opportunity of examining the parts, he found that the area in which the original swelling appeared was occupied by a quantity of yellowish inspissated material which he described as "a dried-up abscess". When the appearance of the calvaria as figured in Smith's paper is recalled (*Fig 564*), there can be little doubt that the case was an example of lipoid granulomatosis. The record is, of course, incomplete, and there are many details about which, in the light of present knowledge, we would like infor-



FIG 564 —The skull of Thomas Smith's case, showing the characteristic calvarial defects. (Reproduced from Smith's original article, published in the *Transactions of the Pathological Society of London*, 1865, Vol. xvi.)

mation, but the evidence which exists suggests that here was an example of the disease with which this paper is concerned.

I have already alluded to Hand's case. He published the details in *The Archives of Pediatrics*, 1893, under the heading "Polyuria and Tuberculosis", and, according to his description, what he regarded as tuberculous deposits in the calvaria and skull base were associated with the symptom of polyuria. Everything points to its having been a case of lipoid granulomatosis manifesting the signs of pituitary involvement.

In September, 1905, Thomas Kay³ presented before the Medical Society of Pennsylvania a boy 7 years of age who showed a clinical picture presenting exophthalmos, polyuria, and extensive defects of the calvaria. Kay expressed the opinion that in this case a lesion existed in or about the floor of the fourth ventricle, but he appears to have made no attempt to explain why a lesion in this situation should be associated with osteoporosis of the bones of the cranial vault.

In 1915 Artur Schuller⁴ described three cases of varying symptomatology, each of which showed defects with irregular outline affecting the membrane bones. A lad of 16 years presented the picture of infantilism, exophthalmos, and the adiposogenital syndrome, a female child of 4 years evidenced stunted growth, exophthalmos, and diabetes insipidus, while in the third case (an adult) there was an infiltration of the skin with yellowish nodules. Schuller did not suggest any common etiological factor in relation to these cases, they were presented in virtue of the radiological interest of the skull defects, but in connection with the girl he ventured an opinion that the skull defects and the diabetes insipidus were caused by a pituitary disturbance, and that the exophthalmos was the result of the pressure of the brain on the orbital contents, being made possible by the large defects in the anterior fossa of the skull, and more particularly in the orbital plates.

Under the heading "Defects in the Membranous Bones, Exophthalmos and Diabetes Insipidus", Henry Christian⁵ published in the *Osler Memorial Volume* of 1919 the report of the case of a girl 5 years old who had been under his care in the Peter Bent Brigham Hospital in Boston. The case presented a typical picture of the symptom complex which the heading implies. In a summary of the detailed case report Christian said, "Diabetes insipidus suggests that the symptom complex is due to a disturbance of pituitary function", and he pointed out that the polyuria was controlled by intramuscular injection of pituitrin. Christian reprinted his original article in the *Medical Clinics of North America*,⁶ and subsequent to his publication the condition which he described came to be known as Schuller-Christian's disease.

In 1921 Hand⁷ encountered a case somewhat similar to that which he had reported in 1893. In this instance a child 3 years old was found to be suffering from exophthalmos, a corneal opacity in each eye (probably the result of exposure), polyuria to the extent of passing 114 oz. of urine daily, and an area of softening in the centre of the right parietal bone. Hand's clinical diagnosis was one of tuberculosis, as it had been in 1893, but on this occasion the child succumbed to an attack of bronchopneumonia, and, as a result of a subsequent autopsy, he advanced the theory that "the primary process might be neoplastic, benign, and myxomatous in character, affecting for some unknown reason the membranous bones and producing exophthalmos and polyuria secondarily by pressure." The pathological report contains a significant statement to the effect that the defect in the centre of the right parietal bone was found to be occupied by a soft yellow substance not unlike caseating material.

The next record with a bearing on the problem is by Thompson, Keegan, and Dunn.⁸ A boy of 9 years of age presented the classical triplex of features—exophthalmos, calvarial defects, and polyuria. The child died, death being attributed to cardiac failure as the result of extensive pulmonary fibrosis. The authors give a detailed account of the autopsy findings, and in describing the condition of the skull they say, "In the centre of the largest membranous areas

(i.e., the calvarial defect) there was yellowish fibrous tissue, and the inner surface of the dura mater was mottled by a yellowish tissue. Microscopical examination of the area showed a lining layer of large oval cells with sharp borders and clear, slightly granular cytoplasm, and small compact centrally placed nuclei. The cytoplasm contained a variable amount of lipoid material in finely divided form." In estimating the cause of the disease, the authors concluded that an infection of some kind existed rather than a metabolic endocrine disturbance, but from the historical point of view the main interest lies in the fact that *this was the first instance in which the existence of lipoid in the lesion was described*.

What appear to have been characteristic cases were reported by Grosh and Stufel⁹ in 1923 (female, 7 years old) and Denzer¹⁰ in 1926 (boy, 4½ years), but these authors had no suggestions to offer regarding the problem of etiology.

Artur Schuller,¹¹ whose contribution to the literature of the disease has already been mentioned, communicated a second paper at the International Congress of Radiology in London in 1925 on what he called "Dysostosis Hypophysaria." He reviewed the cases which had hitherto been reported, and the summary of his conclusions was as follows —

There exists a peculiar type of defect of the skull characterized by the multiplicity, the great size, and the sharp outlines of the defects, localized as well on the cranium as on the base of the skull, combined with defects in the pelvis, exophthalmos, and with some of the symptoms generally diagnosed as due to pituitary dysfunction, namely dwarfism and diabetes insipidus. I propose for this symptom complex the name of Dysostosis Hypophysaria.

It is evident from these remarks that Schuller maintained the view which he expressed ten years before, that the disorder was primarily of pituitary origin.

In 1926 Kyrklund¹² published the case records and the autopsy findings of a 14-year-old-girl who four years before had developed polydipsia and polyuria, and who ultimately succumbed to cardiac weakness. He records characteristic findings in the calvaria and skull base. The hypophysis did not show pathological findings, but there were brownish-yellow deposits in the brain-stem behind the hypophysis. He mentions that the lungs showed extensive changes. Kyrklund's final conclusion was that the deposits were of a sarcomatous nature, and that the changes in the brain-stem behind the hypophysis were responsible for the diabetes insipidus.

In 1927 a case, the clinical findings of which were reported by W. R. Stowe,¹³ came to autopsy, and D. S. Milne has given us an interesting and important account of the pathological findings. I shall quote a portion of his report.

On reflecting the scalp a number of swellings resembling putty in appearance but of fairly firm consistency were observed. These areas were extensions through small perforations in the skull from the dura, which was extensively infiltrated. The brain was normal, a compact yellow mass about the size of a walnut rose up out of the sella turcica, displacing the hypophysis, but without eroding or distorting the clinoid processes. Another mass projected back from the right wing of the sphenoid, which was extended to the ethmoid and both orbits.

Continuing with a report of the histology of the abnormal tissue, Milne states —

The striking yellow mass [was] composed of spindle cells with clear spaces filled with granular yellow substance. Scattered throughout the tumour were giant cells with rosette nucleus surrounded by a granular zone. The nodules from the dura showed the same changes. The entire pituitary was invaded by the yellow growth.

The exact nature of the error appears to have been the source of considerable difference of opinion. Milne came to the conclusion that the condition represented a tumour arising in the hypophysis, Dr Lynch suggested that it might be a chordoma, though as an alternative he put forward the suggestion that the tissue showed many of the characteristics of a xanthoma, and this may be said to be the first hint as to the true nature of the condition.

In 1928 R. S. Rowland,¹¹ of Detroit, published an article in the *Archives of Internal Medicine* which supported Lynch's view of the origin of the disease. The title of the article is "Xanthomatosis and the Reticulo-endothelial System", and in a more elaborate title the contribution is described as "Co-relation of an Unidentified Group of Cases described as Defects in Membranous Bones, Exophthalmos, and Diabetes Insipidus (Christian's Syndrome)". Rowland reported two cases illustrative of the syndrome, one of which came to autopsy, the subjects being both male children, aged 5 years and 3 years and 4 months respectively. Rowland's conclusions on the nature of the condition amounted to this—that he regarded the cases as representing a form of xanthoma in which portions of the reticulo-endothelial system became the site of excessive lipid storage, with resulting hyperplasia of lipid-containing cells. In his own words "One is not dealing with true neoplasm, but with hyperplastic new formation, i.e., lipid storage tumours. The formation of these nodules is a compensatory act on the part of the body in its attempt to rid the blood of an excess of lipid which cannot be properly excreted."

In 1929 Hausman and Bromberg,¹² in reporting a case which had been under treatment in the Bellevue Hospital, New York, on account of pronounced exophthalmos, described the deposits which occur in the region of the hypophysis, and drew attention to the fact that the changes are often relatively widespread, affecting not only the pituitary but also the stalk and the tuber cinereum, and in this connection they raised the interesting possibility that the para-infundibular unit may in some way exercise an atrophic influence upon bone, so that a pathological disturbance of the area may be the factor responsible for the defects in the skull bones.

Since 1931 there have been a number of additional references in literature, but in this survey I have been satisfied with outlining what may be called the milestones in the recognition of the disease, and, for those who may be particularly interested, a moderately complete bibliography is appended (p. 824).

A CONCEPTION OF THE DISEASE

As a result of clinical observation and a certain amount of experimental research, a more or less precise conception of what the disease implies has now been arrived at, and, though there are many points which are still in dispute, we have a fair appreciation of its characters.

Lipoid granulomatosis (and I prefer to employ this collective terminology) is a process by which lipid substances are deposited in selected tissues, with the result that the areas so affected become the sites of granulation tissue formation bearing a close resemblance to malignant tumour, and, in virtue of a destructive influence upon certain organs and tissues, a characteristic syndrome of clinical features is produced. As I have already indicated, there is variation in the type of lipid, and in association with these variations in type different groups of tissues are affected and different sites of deposit selected. It is upon this basis

that we encounter such clinical conditions as Niemann-Pick's disease, in which a lipoid of the lecithin or phosphatid type is deposited in the lymph-vascular and hæmopoietic tissue of the liver, the spleen, and the bone-marrow, Gaucher's disease, in which a lipo-protein of the cerebroside type is deposited in a similar group of tissues, xanthomatosis, in which a cholesterol lipoid appears in relation to epithelium, Tay-Sachs' disease, the essential feature of which is an accumulation of a phosphatid lecithin lipoid in the tissues of the central nervous system, Schuller-Christian's disease, the condition with which this paper is more particularly concerned, and probably others not yet investigated or classified

In this relatively wide field we are concerned with one variety of the disturbance. It is that which is known to the medical profession as Schuller-Christian's syndrome, and its basis is a deposit of cholesterol lipoid in relation to reticulo-endothelial tissue, and more particularly the reticulo-endothelial tissue of membrane bones, serous surfaces, and vascular areas

In order to present a reasoned appreciation of the disease, I propose to summarize certain of the more important facts regarding the two elements which govern and control its development, these elements being the reticulo-endothelial tissues and the cholesterol lipoid

The Reticulo-endothelial Tissues—The recognition of the tissues which comprise the reticulo-endothelial system may be said to depend upon two properties—the morphological peculiarities of the cells as revealed in staining reaction, and the function which the cells appear to provide. All that need be said in respect of the former is that cells in this group show a vital staining reaction when submitted to such a stain as trypan blue, that is to say, they possess the property of absorbing and retaining the granules of the dye within the substance of the cytoplasm, and there is the further fact that the physiological significance of the cell appears to depend upon the degree to which the stain granules are accepted and retained. In describing the distribution of reticulo-endothelial tissues the term 'ubiquitous' might well be applied, for the system includes (1) The endothelium which lines blood- and lymph-vessels, (2) The adult connective-tissue cell or fibrocyte, (3) The cohesive reticulum cell of splenic and lymphoid tissue, (4) The reticulum and endothelial cells which lie in relation to the sinusoids of the spleen, the lymph nodes, the liver, the bone-marrow, the suprarenal cortex, and the hypophysis, (5) The large phagocytic and motile cells of connective tissues (macrophages or histiocytes), (6) The blood monocytes. In this widespread distribution, if we estimate functional importance upon the basis of such a morphological standard as staining reaction, the evidence would seem to indicate that it is the fourth group, the reticulum and endothelial cells lining the sinusoids of the spleen, liver, lymph nodes, bone-marrow, etc., which play the most active part in the physiological activity of the series.

The functions of the system are as yet imperfectly understood, but it seems that cell reproduction, phagocytosis, and certain metabolic processes are probably its most important activities. With the first of these we are not for the moment concerned beyond indicating that the process of cell reproduction or metamorphosis tends to focus itself upon the formation of such active phagocytic elements as large monocytes, histiocytes, and macrophages of the polyblast type. But, for reasons which will presently become apparent, we have special interest in the phagocytic activity of the system. It is evidently a remarkably efficient arrangement, for its

activities are apparent on a variety of tissues—on other cells in the form of red and white blood-corpuscles, muscle fibres, and damaged cells of any type, on such foreign materials as the granules of stains and dyes, on bacteria, and on such metabolic products as hæmoglobin and lipoid. The majority of these various phagocytic activities must be regarded as reactive, but when exercised in relation to products of metabolism may be described as functional, for it is a physiological reaction, a necessary attribute of health. It may seem a misnomer to speak of this last rôle as phagocytic, but in so far as it is a cell inclusion it is such, only in this instance it extends beyond a simple inclusion and destruction, for the inclusion is but a stage in a further metabolic change. Thus we find that hæmoglobin is phagocytosed in preparation for its conversion into bilirubin, while the lipoids, and more especially cholesterol, are phagocytosed for purposes of storage, and no doubt for subsequent release when such is demanded.

In summary, therefore, we may say that the reticulo-endothelial system is a tissue arrangement of widespread and general distribution, that it finds its most efficient arrangements in relation to the blood-sinuses of certain viscera, to bone-marrow, and to serous surfaces, and that among its most striking properties are its phagocytic abilities in conjunction with its power of including, storing, and metabolizing such substances as hæmoglobin and cholesterol.

The Cholesterol Lipoid.—The evidence we possess indicates that an excess of cholesterol in certain body tissues is the primary factor in the production of the clinical syndrome of lipoid granulomatosis (Schuller-Christian's disease), and to make this discussion sufficiently inclusive it is well that we should examine certain of its more important properties and functions. It is one of the large group of fatty substances which, in conjunction with proteins, occurs in the protoplasm of body cells, and to this group the inclusive term 'lipoid' is applied. This word was first introduced in relation to the activity of certain narcotic drugs, for these substances are soluble in lipoids, and, in view of the relatively large percentage of lipoids which exist in nerve-cells, it is believed that it is the alteration in the physical condition of nerve-cells resulting from the solution of the drug in the lipoid which results in narcosis.

According to Cameron,¹⁶ cholesterol is one of the group of simple lipoids, in contrast with the compound or complex lipoids. It may exist in pure form, in which event it is an unsaturated monohydric alcohol, or it may occur as an ester, being combined with a fatty acid as a palmitate, an oleate, or a stearate. Cholesterol and its esters are present in nerve-tissue, in red blood-corpuscles, in bile, in lymphoid tissue, in the spleen, in the adrenal cortex, and in conjunction with fats throughout the body generally. Its functions are not clearly understood, but, according to Wile¹⁷ and his co-workers, cholesterol exerts a protective action, by which body cells are preserved against the influence of such toxins as are liable to enter the body through the avenue of the alimentary canal or of the skin. It exists in the envelope of red blood-corpuscles in order to prevent hæmolytic and destruction of the blood-cells by organisms or their toxins. Under certain pathological conditions it is deposited in appreciable quantities, as when it is encountered in gall-stones, in the atheromatous patches of arteries, in the walls of tuberculous foci, and in carcinomatous tissues.

In speaking of the question of the formation and the metabolism of cholesterol, Cameron, whose views have been already quoted, says —

While normal diet contains cholesterol, or sterols easily convertible into cholesterol, evidence is accumulating that the animal organism can synthesize the compound as need arises, for example, the developing chick shows an increase of cholesterol which can only have been formed by synthesis, a newly-born dog fed on a diet poor in cholesterol for four weeks shows at the end of that time an increased cholesterol twenty times the amount administered in the food

That cholesterol is subject to a certain amount of metabolic change in the body tissues seems to be suggested by the fact that intestinal juice, pancreatic juice, bile, and blood contain an enzyme which will hydrolyse cholesterol esters, while pancreatic juice contains an enzyme which will synthesize the esters from their constituents. During its passage through the intestine a considerable portion of the cholesterol is reduced to coprosterol ($C_{27}H_{47}OH$) by bacterial action.

Such is a summary of the physiology of the two elements, derangements of which constitute the essential pathology of lipoid granulomatosis.

The Constancy and the Origin of the Hypercholesterolaemia—Let us now consider the problem from a local standpoint. When a lipoid granulomatosis error arises, one finding is constant—it is that of an excess of lipoid in the body tissues, and more particularly in the blood and lymph fluids. It has been reported by Christian, Rowland, Sosman, and others, and of the three cases which came under personal observation the two which were investigated from this point of view showed a definite cholesterolaemia. There seems to be no doubt that this factor is the original and essential feature underlying the local manifestation, and such being the case, it may be that an attempt on the part of certain body tissues to eliminate a portion of the excess results in the individual pathology. It is therefore reasonable to inquire as to the factors which control lipoid metabolism, for it is by an understanding of these that we are likely to arrive at a knowledge of the conditions which are responsible for the increase which characterizes the disease.

Raab¹⁸ claims that the process is under the control of the autonomic nervous system, initiated in the pituitary gland, whence impulses are relayed by the autonomic centre in the floor of the third ventricle and the hypothalamus to the sympathetic fibres, which in turn act as a mobilizer of stored lipoids, at the same time promoting the destruction of any excess in the liver. This theory is of peculiar interest in relation to the subject under discussion, for there is a stage in the progress of the disease in which there is evident derangement of the pituitary gland and the hypothalamus, but, as we shall see, the pituitary-hypothalamic involvement is a relatively late stage in the pathological sequence, so that any influence which such changes may exercise can only be secondary in their effect. While there is no evidence of a primary pituitary disturbance, it may be, if Raab's views are correct, that the changes which are ultimately wrought upon the gland in lipoid granulomatosis are a factor in maintaining a hypercholesterolaemia.

I believe I am correct when I say that, according to the views of the majority of biochemists, the liver and the lungs are the visceral areas in which the cholesterol balance is maintained, and in support of this contention the significant fact is quoted that blood from the portal vein contains a larger proportion of cholesterol than that from the hepatic veins, while similarly the percentage in the pulmonary artery exceeds that found in the pulmonary veins. If we apply the truth of this finding (Cameron) we may argue that the factor responsible for the occurrence of hypercholesterolaemia is a disturbance of the tissue arrangements in the liver and

in the lungs, so that either an excess of lipid is produced or the process of destruction which maintains the balance is interfered with. At first sight it might seem that an increased ingestion of lipoids offers the most likely explanation of a body excess. A great deal of experimental work has been done on this aspect of the question, the results are by no means conclusive, but the fact emerges that, as soon as an increased intake is established, that which constitutes an excess is dealt with by cells of the reticulo-endothelial system. So far as I am aware, the position is that, while an increase of the lipid ingestion may result in a temporary excess of lipid in the body fluids, the balance is quickly adjusted through the medium of the reticulo-endothelial system, and it is impossible to institute a hypercholesterolaemia for any length of time. Experimental proofs of this truth are numerous. Amtschkow and Chalutow¹⁹ fed rabbits on a diet rich in cholesterol, after some time the histiocytes of the spleen and lymph nodes were found to be loaded with the lipid, but no appreciable increase was apparent in the blood or lymph fluid.

Working on the same lines, Yuasa²⁰ obtained very similar results. He employed omnivora in his experiments, because he felt that in this instance cholesterol would not constitute a foreign article of diet, as it might if herbivora were used.

Fontana²¹ and his colleagues fed rabbits on large amounts of viosterol. They confirmed the results of previous observers in regard to the response shown by reticulo-endothelial cells, and in more than one instance the cell reaction in the histiocytes of the spleen was so pronounced as to result in a definite enlargement of that organ with a histology very similar to that which is encountered in the lipid granulomatosis of Niemann-Pick's disease. The experiments do not record any coincident increase of lipid in the circulating body fluids.

These various observations have brought out one feature of much practical significance, which is fully elaborated by Chalutow²² in his most recent paper, and it is this—that when lipid deposits are produced in the body tissue by such experimental means as feeding animals on an excess lipid diet, the deposits appear to arise simultaneously and suddenly in what might be described as a shower (*Springartige*), an evidence which suggests that the body possesses a threshold to lipoids, as it does to other substances. If the threshold is passed, the substance becomes foreign to the body and is dealt with by a widespread process of inclusion and subsequent destruction.

If we sum up the experimental evidence in so far as it helps to illuminate the problem of hypercholesterolaemia, the findings indicate that the excess of lipid in circulation probably depends upon a failure on the part of the liver and lung tissue to maintain the body lipid at a normal level, as a result the reticulo-endothelial tissues attempt to eliminate the excess by a process of absorption and deposit, and, according to Chalutow, the deposit is a multiple and simultaneous one accomplished as soon as the physiological threshold is exceeded.

THE ORIGIN OF LIPOID GRANULOMATOSIS

In attempting to offer an explanation of the process which results in the deposit of localized collections of cholesterol so as to constitute the clinical condition of lipid granulomatosis (Schuller-Christian's disease) there must be a considerable element of hypothesis. Bearing this in mind, it is suggested that

the following is the outline of events, as we suppose they developed in the cases which have been described

The initial change is one of increased lipid (cholesterol) content in the circulating body fluids. The possible explanations of this have been discussed, and, though there is no certainty as to the exact nature of the elements which initiate the disorder, there is reason to suppose that it originates in some change (possibly prenatal) occurring in the areas which control lipid (cholesterol) metabolism—the liver and the lungs. As a result of the lipid increase, and in an attempt to adjust the balance, the reticulo-endothelial tissues of certain body areas proceed to concern themselves with absorbing and depositing the substance so that accumulation occurs in individual areas. This phase is not accomplished without considerable cell reaction among the tissues concerned, and the changes which ensue may be said to constitute the characteristic pathology of the lesion.

MORBID ANATOMY AND HISTOPATHOLOGY

The morbid anatomy of the fully established disease may be said to consist in the deposit in certain areas of a granulation tissue of characteristic appearance. The collections are firm in consistency and putty-like in appearance, in certain instances, apparently where the lesion has been longest in existence, the tissue may assume a yellowish coloration.

Deposits of this nature may be encountered in a variety of situations, and from our own observation, together with a study of the literature, the following sites have been recognized: the sub-calvarial dura, whence it extends into the diploe,

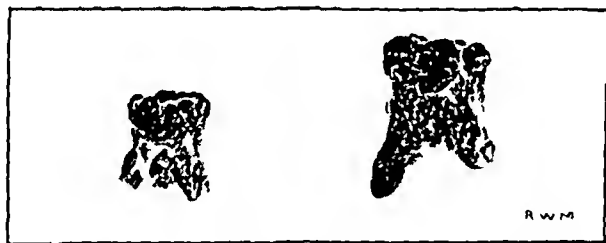


FIG 565—Case 3 Two teeth from the mandible. Note the lipid deposit which covers the roots and the bodies.

probably along the line of an emissary vein, and ultimately destroys large areas of the bone, the dura of the skull base, from which area there is extension into the sella turcica, and through the superior orbital fissure into the orbit, the cancellous centre of the lower jaw, in which event it possibly is an extension from the vascular tissues at the apices of the teeth (Fig 565), the mastoid air-sinus, the clavicle, the pelvic bones, the ribs, the vertebræ and the long bones, the cervical lymphatic nodes, the pleuræ and the lungs, the liver, where the change has been noted in relation to Kupffer's cells, the macula, and the left cerebellar hemisphere (Cushing). Microscopic lesions suggestive of the pathology have been described in the stomach wall (Davidson²³), but nothing abnormal has been noted in the suprarenals, the pancreas, or the thyroid.

It is evident from this summary that the distribution of the deposits is widespread, but it is also apparent that there are certain sites of selection, and that these are the mesothelial areas of the basal and calvarial dura and the pleura. Why this selection should be exercised is difficult to explain, unless it be that the reticulo-endothelial tissues of these areas possess some special function in dealing with lipoid excess. One might anticipate that the spleen would form such an area, but, as far as I am aware, no evidence of its selection has been encountered.

The various cytological changes which underlie the development of the granulation tissue have been made the subject of detailed observation, and the phases have been described by Sosman²¹ and others. The process seems to

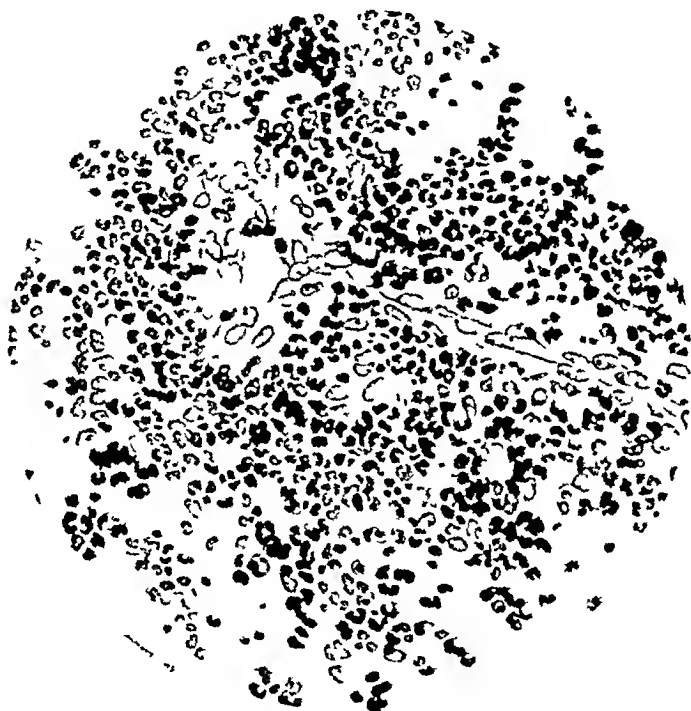


FIG 566 —Drawing of a section showing an early phase of the pathology. The endothelial cells lining a capillary have become swollen and detached from the vessel wall. A certain number have begun to migrate into the surrounding tissues. Many of the cells show a commencing lipoid accumulation in the cytoplasm. ($\times 300$)

be as follows. Recognizing the essential initial feature that the body fluids, and the blood in particular, contain an excess of cholesterol content, in one area characterized by a profuse capillary circulation there is a blockage of a capillary lumen as a result of swelling and proliferation of its lining endothelium (*Fig 566*). It is not clear why this change should occur, it has been suggested that it is due to local injury, but on the other hand it may be that it is the result of a cholesterol concentration acting as an irritant. Whatever the cause, the endothelial swelling and proliferation are such as to constitute a local barrier to the capillary circulation, and from this centre the subsequent pathological changes spread and develop. That an irritant process of some kind is at work is suggested by the succeeding phase, for around the obstructed area an intense concentration of lymphocytes and

eosinophilic leucocytes becomes evident (*Fig 567*) In the meantime the capillary area loses all trace of its tubular structure, and the point at which the original changes appeared is occupied by a number of oval and fusiform cells which in respect of their staining reaction show evidences of their vascular origin Scattered among the mononuclear cells are giant cells of a multinuclear type which have obviously arisen by a syncytial process Lipoid deposits now become evident in the cell cytoplasm (*Fig 568*) The lipoid is first recognized in the mononuclear cell which has been derived from the vascular endothelium, and its appearance synchronizes with an interesting development, for the lipoid-containing cells proceed to congregate in syncytial-like masses so as to constitute the so-called multinuclear giant cells of the lesion (*Fig 569*) A stage is eventually reached in which the lipoid-laden cells either discharge their contents or lose their definition as cells, and become amalgamated with a mass of lipoid and cellular debris In so far as the cell reaction is concerned, interesting individual facts emerge, two of which are that the lipoid, when it enters the cell, collects in the perinuclear area, and that cells so affected cease to show any sign of nuclear division (*Fig 570*) The latter is a remarkable feature, it has been pointed out by several observers, and it cannot fail to arouse interest that a tissue which shows wide variation in cell structure and type should be characterized by a relative absence of nuclear activity

Sosman has pointed out that the cellular arrangements appear to follow an interesting cycle He observed that the more mature type of foam or xanthoma cell is found towards the periphery, while the small mononuclear and evidently younger class of cell occupies the centre of the lesion—the area, in other words, in which the vessel, the real centre of the disturbance, originally lay One is left with the impression that the process is one of flow and ebb, the immature cell passing inwards to receive its cholesterol content, and, having done so, passing to the periphery laden with its intracellular burden

These are the cellular arrangements which constitute the characteristic granulomatous tissue deposit, but as the lesion develops there is a natural tendency towards fibrosis The cells which previously acted as macrophages become converted into fibroblasts, with the result that the tissue acquires a curious nodular character, an indiscriminate arrangement of fibrous tissue, giant cells, fibroblasts, and lipoid deposits which are partly intracellular and partly free

Such are the phases of cell activity in the lesion as the microscope reveals them, and I think all will agree that the picture is a remarkable demonstration of the effects which follow the ingestion by the cell of what is usually regarded as a chemically inert substance Bayliss,²⁰ in his *Principles of General Physiology*, suggests that the ubiquitous distribution of cholesterol indicates that it plays some important part in the mechanism of the cell, but he does not state what that influence may be As we view it, the appearances suggest that its existence in any quantity results within the body in some degree of irritation—its presence at least calls into action the great phagocytic function of the reticulo-endothelium, and what is regarded as granulation tissue is but the response of phagocytic elements in their attempt to deal with the invasion One of the most impressive features in the histopathology is the arrest of karyokinesis, there appears to be no nuclear division in the cholesterol-containing cell, and it may be a significant fact that when the lipoid enters the cell it appears to collect in the perinuclear area Can it be that its effect in bulk is to arrest nuclear division, and that in amounts which are

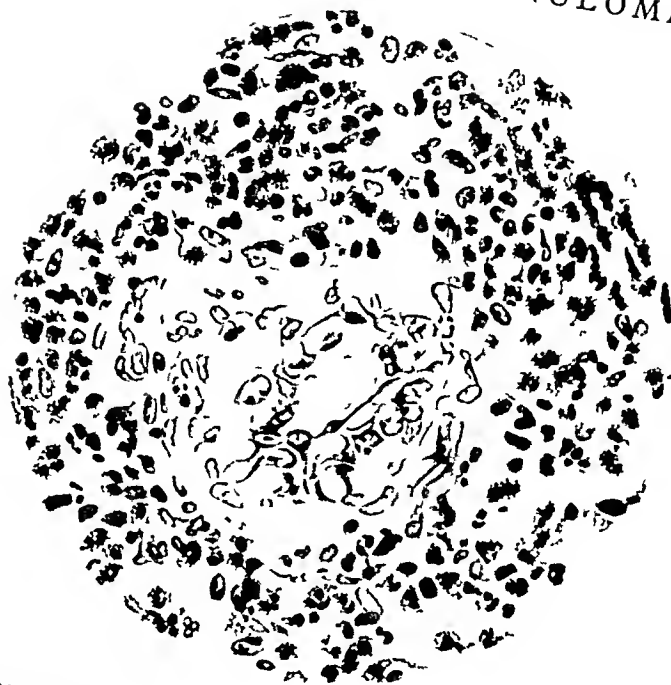


FIG 567—Drawing of a section showing the early stage of lipoid granulomatosis. A capillary in cross section shows the swelling and distortion of the lining endothelial cells. An intracellular accumulation of lipoid is apparent in certain cells. Note the accumulation of lymphocytes and eosinophils in the perivascular area (445)



FIG 568—Drawing of a section showing the stage of lipoid deposit. The original outline of the capillary is shown. In the centre of the field there is a deposit of lipoid, while a number of lipoid laden cells which have not yet discharged their contents are seen around the periphery (280)

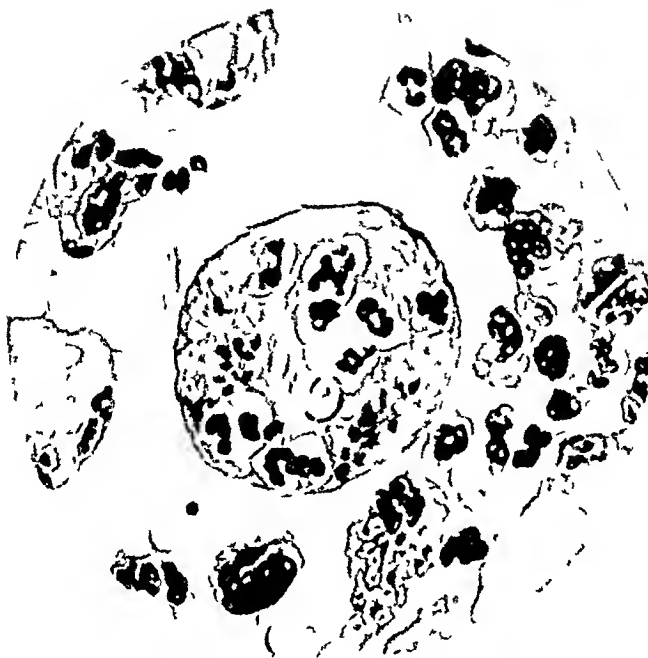


FIG 569 —Drawing of a section showing the structure of the so called multinuclear giant cell. The appearance is produced by the fusion of a number of individual lipid-laden cells. Note the fashion in which the lipid appears to collect in the perinuclear area. The majority of the surrounding cells are lipid laden histiocytes and reticulum cells. (630)



FIG 570 —Drawing of a section giving an impression of the characteristic cytology of lipid granulomatosis. There are multinuclear giant cells (syncytial masses) showing lipid deposit in the perinuclear areas, lipid-laden histiocytes, and reticulum cells. There are also collections of structureless lipid. (x 255)

in keeping with health, it has the effect of controlling cell division.² One appreciates that, if this theory could be substantiated, lipoidosis might conceivably have an important bearing upon the influence of the growing tissues, even neoplastic, where mitosis is abundant.

The Effect of the Granulation Tissue Formation.—The formation of the granulation element is associated with certain untoward results. Its ability to infiltrate is insignificant, but it spreads with relative ease in parts occupied by areolar tissue, in such fat-containing areas as the orbit, and in the perivascular spaces. What appears to be an infiltration of an organ is an extension along perivascular channels, and it is evident that fibrous tissue presents an impenetrable barrier to its invasion. Its progress in bone is marked by a progressive and extensive decalcification of trabecular and later of compact bone, with the result that large portions of the calvaria, clavicles, or mandible (all more or less membrane bones) may

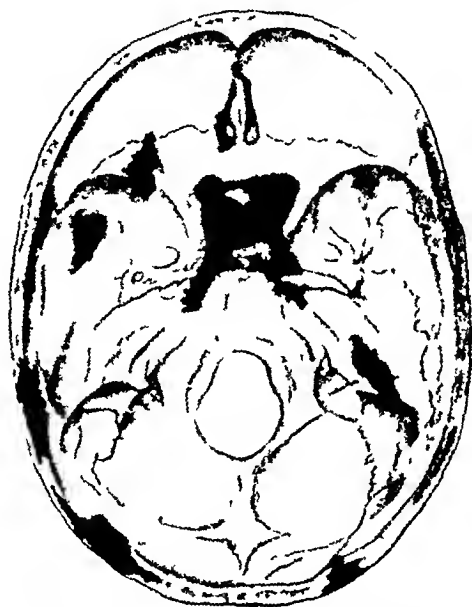


FIG 571 —The skull base, illustrating the distribution of the granulation tissue deposit in the pituitary fossa, extending along the optic nerves to the orbits, and eventually to the lamina cribrosa of the ethmoid bone.

disappear, and from what we find it seems that the process of bone removal is one of osteolysis or phagocytosis initiated and accomplished by the larger type of multinuclear giant cell, which is universally found at the periphery of the lesions. It is significant that, though large areas of bone may be involved, and the skull vault reduced to a condition which has been described as gelatinous, the underlying dura and the overlying scalp often remain unaffected. Deposits in the basal subdural area are rare except in the space which lies around the hypophysis (*Fig 571*). This area is so frequently affected that it may be regarded as a site of election, and in some instances the sella turcica is filled with a putty-like mass, with the result that there is displacement of the hypophysis and traction or pressure on its stalk. In a few instances infiltration of the hypophysis and of the hypothalamic area has been described, but such is rare. It is a remarkable feature that, though there may be pronounced accumulation in the sella turcica, there is no invasion of the

basil-sphenoid or of the clinoid processes. From the hypophysial fossa the granulation tissue extends forwards around the carotid vessels, and thence in the tissue around the optic nerve, until it finds itself in the orbit, where factors of space and of fat content permit such accumulation of the deposit that the eyeball is displaced.

The effect of the disturbance in regions other than bone is a displacement process, surrounding elements are pushed aside, compressed, and atrophied, tissue spaces are filled and distorted, but, so far as can be observed, it is only in relation to bone that one witnesses the process of phagocytosis which so intimately affects the skeletal outline.

I need not elaborate the pathology further, for the process is a reproduction in different tissues of the group of changes I have outlined, and, but for variation in situation, there is no difference or distinction between the lesions wherever they may occur.

The summary of the pathology is briefly that in certain tissue areas there is a formation of granulation tissue of distinctive cytology, the result of reactive changes in reticulo-endothelial tissue, the process being a reaction to a cholesterol excess in the blood and body fluids.

THE CLINICAL FEATURES

Various observers have described the disease as being distinguished by a trilogy of clinical features—diabetes insipidus, exophthalmos, and calvarial osseous defects. It is true that these are the most obvious and constant evidences of the disease, but there are variations in the clinical picture, and it is probably more accurate to discuss the clinical evidences on a pathological basis.

Children who are affected by the disease are invariably poorly developed and underweight. The obvious explanation is that metabolism is deranged and the processes of tissue nutrition are upset, but to stop there is to leave the real problem unanswered. There must be some original factor underlying the metabolic disturbance. It has been suggested that as a result of displacement of, and pressure upon, the pituitary, growth functions are inhibited and delayed, or that the hypercholesterolaemia in some way affects nuclear division so that the whole process of tissue growth is retarded. The second evidence of the disease concerns the mental attitude of the child. He is cross, irritable, and most difficult to examine. He cries a great deal, and his mental balance appears to be easily upset. I believe that these changes have an evident explanation. Early in the disease there occurs an epidural formation of granulation tissue, and it seems likely that as a result of such a deposit there is an increase of intracranial tension causing discomfort, pain, restlessness, and irritability. In the intervals of rest the child appears apathetic, with an open mouth and a somewhat listless expression. A further evidence of increasing intracranial tension is seen in the condition of the scalp veins. They are unduly prominent, because the epidural infiltration tends to interfere with the natural evacuation of intracranial venous blood by the basal foramina and orbital fissures, and, in order to compensate for this, the scalp veins are engorged and large.

As soon as the peri-hypophysial infiltration becomes established (and it is at a relatively early stage in the progress of the disease) some of the most characteristic features make their appearance. It will be recalled that the arrangement of the

dura mater in the sella turcica consists of two layers, a periosteal and a visceral, the former adhering to the bone, the latter being raised to form the diaphragma sellæ like a tent over the clinoid processes. Between these layers lies a space occupied by the cavernous sinuses and the pituitary body, these structures being separated from each other by endothelial membranes. It is within this endothelial area that the granulation deposit occurs, and as it increases, it is evident that pressure, tension, and traction influences may be exerted not only upon the pituitary but upon "the pituitary complex"—the pituitary proper, its stalk, and the adjacent hypothalamic region of the brain. The clinical evidence of these disturbing influences is shown in two manifestations—the polydipsia and polyuria of diabetes insipidus, and an infantilism sometimes combined with the adiposo-genital syndrome. The diabetes insipidus manifestation is the more constant, and in respect of priority of symptoms, the first to appear. It is probably due to irritation of the hypothalamic region, the result of tension upon the stalk of the pituitary. Similar symptoms have been produced by experiment and in association with pathological evidence (Greung²⁶), and it is more reasonable to accept this explanation than to assume that they proceed from influence exerted upon and restricted to the posterior lobe of the pituitary. The infantilism and genital syndrome are a later development, and there seems no reason to doubt that these changes arise from disturbances of the anterior lobe, the structure of which is such that perivascular invasion by the granulation tissue may occur.

The explanation of the exophthalmos is apparent. From the region of the pituitary fossa the granulation tissue extends forwards along the endothelial membrane which separates the cavernous sinuses from the pituitary body, it surrounds the optic chiasma, and, passing through the optic foramen and superior orbital fissure, it reaches the orbit, where it accumulates to such an extent that exophthalmos may result. The degree of the disturbance varies—in some cases it is scarcely perceptible, in others (as in a case recorded by Wheeler) the protrusion is so pronounced that it may become necessary to close the palpebral fissure artificially in order to protect the globe.

The calvarial osseous defects have been described in relation to the pathology, and I need not elaborate this point further beyond repeating that the granulation tissue begins in the epidural or intradural area whence it extends into the diploe, probably along the line of entering vessels, for the most constant sites of the original deposits in the bones are at those points in the parietal and occipital bones at which such vessels are encountered. In resistant or untreated cases the calvarial destruction is so complete that the condition fully justifies the description which the French have used—the term 'gelatinous skull'. As I have already indicated, deposits may occur in the clavicles, and they are encountered, though less frequently, within the long bones.

While diabetes insipidus, exophthalmos, and cranial deficiencies may be said to constitute the classical trilogy of clinical features, other evidences may arise according to the area in which the granuloma occurs, and theoretically the changes may appear wherever reticulo-endothelial tissue is encountered. There may be lymph-node enlargement from deposits in lymph-vascular tissue, jaundice may arise from changes relating to the distribution of Kupffer's cells in the liver, there may be signs suggestive of pleurisy or even of pulmonary consolidation, the result of changes in the pleura or in the cells of the alveoli. It comes to this, that the

clinical features are direct manifestations of the pathology, and, in view of the widespread distribution of the tissues which provide the changes, the possible variation in signs and symptoms is considerable

DIAGNOSIS

As a rule the diagnosis does not present particular difficulty. The disease is no doubt comparatively uncommon, but cases probably occur more frequently than we realize. They are confused with tuberculosis, with tumours of bone, and with primary pituitary disease. Literature records at least one instance in which a child underwent removal of both eyes because those in charge of the case ascribed the exophthalmos to a sarcoma involving the orbital cavities.

In early cases the diagnosis is based upon the following points—the age and general appearance of the child, the occurrence of defects in the calvaria, accompanied, it may be, by the presence of scalp swellings, the development of polyuria and polydipsia, the result of pituitary involvement. In late cases there is dwarfism, exophthalmos, and the adiposo-genital syndrome. The radiographic appearance of the skull bones is often characteristic, the lacunæ showing clear-cut edges with irregular outlines, so that the skull defects may present a map-like or geographical outline, to which the Germans give the name '*Landkartartige*'. The blood readings show an excess of cholesterol, of total acids, and of lecithin. The characteristic histological picture is a proliferation of the reticulo-endothelial cell groups illustrating the presence of large numbers of phagocytic cells, multinucleated giant cells, and the large lipid-containing or foam cells.

The cytology of the area is characteristic, but here too there are possibilities of confusion, for the picture may be mistaken for that presented by the simple giant-cell tumour, by a myeloma, by tuberculosis, and by syphilis.

PROGNOSIS

The prognosis in large measure is dependent upon the stage at which the disease comes under treatment, but in general it may be said that, while there are no doubt variations in the degree of acuteness of the disturbance, the general tendency is towards an increasing distribution and extent of the disease. A patient referred to by Christian²⁷ was alive and relatively well at the end of ten years, Schuller's²⁸ patient was well at the end of sixteen years, one of Sosman's²⁹ patients enjoyed relatively good health for seventeen years. Rowland³⁰ collected the results of 14 cases in 1928, of which 7 individuals had died of the disease, while 7 were alive. Sosman³¹ reported 9 additional cases in 1931, with 7 survivals. It is our own experience that, if the condition is recognized early, and if appropriate treatment is instituted, the prognosis is relatively good. Of the 3 children who have come under our observation, 2 are alive and well, one at the end of five years, while the third succumbed to an intercurrent infection. It is possible that even in the absence of treatment a tolerance is acquired, or the lesions heal and disappear.

Those who pass to a fatal issue because they have been unrecognized, or because they have failed to respond to treatment, succumb for various reasons—it may be from asthenia and general weakness, or heart failure, or the development of an intercurrent disease reacting on a system which is already debilitated to a serious extent.

TREATMENT

In regard to the question of treatment, certain accepted lines are generally followed. A diet is prescribed which is as far as possible cholesterol-free, but which contains a large percentage of green vegetables. Endocrine therapy in the shape of extract of pituitary is employed, and there is no doubt that this substance controls the diabetes insipidus. Small amounts of thyroid extract were used by Rowland, but apparently without benefit. Irradiated foods, parathyroid gland extract, heliotherapy, cod-liver oil, and calcium have all been tried without success. Sosman has used insulin in the treatment of these cases, giving a dosage of 10 units daily, and he finds that, while it causes improvement in the general health, it apparently has no effect upon the hypercholesterolaemia, nor does it directly influence the granuloma.

The sheet anchor of treatment is deep radiotherapy, and there is no doubt that since this procedure has been followed the results, both immediate and late, have been vastly improved. In our own cases the procedure has been to irradiate the various areas with a dosage of 150 kilowatts 4 milliamperes, each area being irradiated for a period of ten minutes on every third day, a filter of 3 mm of aluminium being used. The total number of applications varies according to the response—in the last case a series of forty has already been given. The effect of the deep radiotherapy is to destroy the distended and lipid-laden histiocytes. The lipid being thus liberated, is disposed of elsewhere, while the original lesion undergoes fibrosis, recalcification, and healing.

SUMMARY

In this paper I have attempted to outline the pathology and the clinical aspects of a significant and interesting disease. There are indications that its incidence is more common than we realize, and the likelihood is that the condition is either overlooked or is misdiagnosed. Perhaps its most outstanding feature is the production of a granulomatous tissue which bears certain resemblances to a malignant tumour, but which arises as the reaction of reticulo-endothelial tissues to the stimulus of a lipid which is excessive in amount, and which possibly possesses abnormal characteristics. The entire clinical picture is dependent upon disturbance of body tissue and function as the result of the pressure and displacement influences of the granulation deposit.

While the prognosis is always serious, there is evidence that by the use of deep radiotherapy the disease can be checked and even permanently arrested.

In the preparation of this paper I have had a great deal of assistance from Mr David M Greig, F R C S E, LL D, Curator of the Museum of the Royal College of Surgeons, Edinburgh. It was he who first drew my attention to the significance of the condition, and he has given me great assistance with the literature, and with many problems of the pathology. I am grateful to Professor Woodburn Morrison and to Dr Leila Hawksley for permission to quote a case which has been under their care, they have also allowed me to make use of certain radiograms and photomicrographs illustrative of the case. Mr John Bruce, F R C S E, has assisted me in the general preparation of the paper. I am indebted to

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REFERENCES

- ¹ HAND, A, *Arch of Pediat*, 1893, 1, 673
- ² SMITH, T, *Trans Pathol Soc Lond*, 1865, 16, 224
- ³ KAY, T W, *Pennsyl Med Jour*, 1905-6, 1, 520
- ⁴ SCHULLER, A, *Forts a d Geb d Rontgenstrahlen*, 1915-6, 1111, 12
- ⁵ CHRISTIAN, H A, *Contrib Med and Biol Research*, 1919, 1, 390
- ⁶ CHRISTIAN, H A, *Med Clin N Amer*, 1920, 11, 847
- ⁷ HAND, A, *Amer Jour Med Sci*, 1921, 111, 509
- ⁸ THOMPSON, C O, KEEGAN, J J, and DUNN, A D, *Arch of Internal Med*, 1925, 111, 650
- ⁹ GROSH, L C, and STIFEL, J L, *Ibid*, 1923, 111, 76
- ¹⁰ DENZER, B S, *Amer Jour Dis Child*, 1926, 111, 480
- ¹¹ SCHULLER, A, *Brit Jour Radiol*, 1926, 111, 156
- ¹² KYRKLUND, R, *Zeits f Kinderheilk*, 1926, 11, 56
- ¹³ STOWE, W R, *Trans Austral Med Cong*, 1927, Sept, 144
- ¹⁴ ROWLAND, R S, *Arch of Internal Med*, 1928, 111, 611
- ¹⁵ HAUSMAN, L, and BROMBERG, W, *Arch Neurol and Psychiat*, 1929, 11, 1402
- ¹⁶ CAMERON, A T, *Text-Book of Biochemistry*, 3rd ed
- ¹⁷ WILE, U J, ECKSTEIN, H C, and CURTIS, A C, *Arch Dermatol and Syph*, 1929, 11, 35
- ¹⁸ RAAB, W, *Zeits f d ges exper Med*, 1928, 111, 366
- ¹⁹ ANITSCHKOW, N, and CHALATOW, S, *Centralb f allg Pathol u pathol Anat*, 1913, 111, 1
- ²⁰ YUASA, D, *Beitr z pathol Anat u z allg Pathol*, 1928, 111, 570
- ²¹ FONTANA, P, MIMILLA, A, and COLLAGO, J A, *Anu de Fac de Med Montevideo*, 1929, 111, 1075
- ²² CHALATOW, S, *Virchow's Arch*, 1929, 111, 691
- ²³ DAVIDSON, J, TURNER, A L, and WHITE, A C, *Edin Med Jour*, 1925, 111, 153
- ²⁴ SOSMAN, M, *Jour Amer Med Assoc*, 1932, 111, 110
- ²⁵ BAYLISS, W M, *Principles of General Physiology*, 4th ed, 134
- ²⁶ GREUING, R, *Die Lebensnerven*, 1924, 78 (edited by R Müller, Berlin)
- ²⁷ CHRISTIAN, H A, *Med Clin N Amer*, 1920, 11, 847
- ²⁸ SCHULLER, A, and CHIARI, H, *Wien klin Woch*, 1930, 111, 153
- ²⁹ SOSMAN, M, *Loc sup cit*
- ³⁰ ROWLAND, R S, *Arch of Internal Med*, 1928, 111, 611
- ³¹ SOSMAN, M, *Amer Jour Roentgenol*, 1930, 111, 581

AN UNUSUAL CASE OF BONE REGENERATION AFTER COMPLETE DIAPHYSECTOMY ON TWO OCCASIONS

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REGENERATION of the diaphysis of a bone such as the tibia on two occasions after the shafts (the original and the new) had been removed on account of acute disease is unusual and worthy of record. More particularly is this so when the new shafts formed resembled the normal bone in size and shape.

The patient—a boy of 6 years—gave a history of having fallen heavily on the right leg three days before coming into hospital. The day before admission he was fevered, and had severe pain and tenderness in the leg. On examination the right leg was swollen and oedematous, there was much tenderness at the junction of the middle and lower thirds of the tibia, and the child appeared acutely ill. At operation a diffuse osteomyelitis and extensive necrosis were found in the tibial diaphysis, which was removed completely. The opening in the periosteum through which the bone was removed was closed with sutures. The shaft removed subperiosteally was completely necrotic, and scattered irregularly over the surface were patches of congestion (*Fig 572*). Where the shaft had been in apposition with the epiphysal plates at the upper and lower ends there was some irregular absorption of bone, at the lower end this absorption formed a dome-shaped depression. Radiological examination four days afterwards showed complete absence of the tibial diaphysis (*Fig 573*), six weeks later there was extensive formation of new bone (*Fig 574*). The child was able to walk six months after operation, and radiologically the bone, though somewhat irregular in outline, appeared to have completely re-formed (*Fig 575*).

The boy remained in good health for two and a half years after dismissal, when he was readmitted complaining of pain over the scar below the right tibial tuberosity. There was a history of slight trauma in this region some days previously. On an incision being made through the old scar, a large quantity of sanguineous pus was evacuated from under the periosteum, and carious bone was removed from the medullary cavity. Four days later there was much swelling in the region of the knee, and the child was gravely ill. An incision was made down the front of the tibia from knee to ankle, and on opening the periosteum an extensive osteitis of the whole shaft was found. To facilitate removal of the bone, the shaft was cut through below the middle, and both upper and lower parts of the diaphysis were excised.

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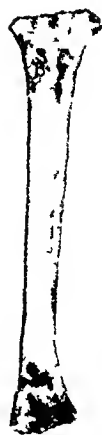


FIG 572—Posterior surface of original shaft, irregular absorption of bone at upper and lower ends (f f)



FIG 573—X-ray plate four days after complete removal of tibial diaphysis



FIG 574—X-ray plate six weeks after operation partial regeneration of tibial diaphysis



FIG 575—X-ray plate six months after operation complete regeneration of tibial diaphysis

This second shaft had developed from the periosteum stripped from the original shaft removed three years previously, and though extensively necrotic, was somewhat remarkable (Figs 576, 577). It was 4.5 cm longer than the original and also thicker, while the various markings were more prominent, and in spite of some irregularity the new bone showed the general anatomical features of the normal bone for a child of this age, even to the position of the foramen for the nutrient artery at the junction of the upper and middle thirds of the posterior surface. Thus growth of the bone corresponding to the general growth of the child had taken place.

A large area of necrotic bone had been removed surgically at the upper end of the medial aspect, where the line of separation from the epiphysial plate was very



FIG 576



FIG 577



FIG 578

FIG 576—Posterior surface of second shaft. The bone is thicker than normal and more irregular in outline. The markings for the muscular insertions are fairly well defined and the foramen for the nutrient artery is present at the junction of the upper and middle thirds. The bone was divided transversely near the lower end to allow of easier removal. ($\times 1\frac{1}{2}$)

FIG 577—Medial aspect of second shaft showing area of necrosis below medial condyle. ($\times 1\frac{1}{2}$)

FIG 578—Anterior aspect of third shaft after amputation. The large abscess cavity extending into the head of the tibia is seen. ($\times 1\frac{1}{2}$)

irregular (Fig 577). A perforation was present in the upper epiphysial plate, and through this infection had reached the knee-joint. The whole wound was thoroughly dressed, and the knee-joint washed out with eusol and a drain inserted. Repeated dressings were necessary during the month following the operation, at the end of which radiological examination showed new bone formation. During the next month the condition of the knee-joint did not improve, the temperature remained high, and the child's condition became more serious. Consequently, it was considered advisable to amputate the leg. Dissection of the specimen after amputation showed an irregular perforation in the head of the bone leading to the knee-joint, in which there was an extensive purulent arthritis, with destruction of cartilage and

synovial membrane The tibial diaphysis had partly regrown (*Fig 578*) and consisted for the most part of osteoid tissue, which showed slight superficial sepsis This third shaft had formed in ten weeks after removal of the second After the amputation the child's condition quickly improved, and he was able to go about on crutches at the end of six weeks

DISCUSSION

Opinion is divided as to the advisability of complete removal of a necrotic diaphysis in cases of acute osteomyelitis In the case reported complete restoration of the part justified the removal of the whole of the dead shaft This, however, may not always occur, and probably depends on the acuteness of the infection The drawing together of the periosteum near the upper epiphysial line was probably a technical error, as it allowed an unavoidable pocket to remain, in which latent infection persisted owing to lack of efficient drainage and gave trouble at a later date Owing to the great vegetative capacity of the osteogenic tissue in the early years of life the diaphysis in this case was on two occasions restored, in the first instance completely, and in the second only partially Further, the new bone in the first replacement corresponded closely in its structural characters, size, and shape to that in the normal child This was probably due to the limiting action of the periosteum, together with the moulding effect of the surrounding muscles

SUMMARY

A short account is given of a case of osteomyelitis of the tibia, with regeneration of the shaft after removal of the original bone and also after excision of the new shaft in which osteomyelitis recurred

THE TREATMENT OF SPASMODIC DYSPHAGIA BY SYMPATHETIC DENERVATION*

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DR D R PATERSON, of Cardiff, was one of the first, if not the first, to describe a now well-known syndrome occurring in women and comprising spasmodic dysphagia, anæmia, and atrophic changes in certain mucous membranes, and to point out the highly significant fact that there is a tendency for patients with this condition ultimately to develop pharyngeal carcinoma. In 1919 Paterson reported his observations, which had been made during the course of many years, to the Laryngological Section of the Royal Society of Medicine.¹

Hitherto the treatment of this condition of spasmodic dysphagia has been largely symptomatic,—exhibition of iron for the anæmia, dilatation of the resistant sphincter at the lower end of the pharynx, and surgical or radium treatment for carcinoma when this supervened.

In discussing the syndrome with my colleague, Mr R D Owen,² who each year sees a number of these cases in his Laryngological Department, it occurred to me that the condition might be alleviated and the possible onset of carcinoma prevented if two desiderata could be established—namely, relaxation of the sphincter (supracæophageal sphincter of Sir William Turner, horizontal part of inferior constrictor, crico-pharyngeus), and secondly, an increase in the blood-supply to the hypopharyngeal mucosa, examination having repeatedly shown that an anæmic and atrophic condition of this mucosa existed in association with tight constriction of the sphincter. Observation had further shown that as these cases progressed the mucosa became damaged by the passage of food and instruments, and changed in character† until ultimately metaplasia resulted in the terminal event, hypopharyngeal carcinoma.

It appeared that we might be able to relax the sphincter and improve the blood-supply if we could remove the sympathetic innervation of the sphincter and the lower part of the pharynx, and investigation showed that the sympathetic component of the pharyngeal plexus was derived entirely from the superior cervical ganglion. It seemed, therefore, logical to perform bilateral superior cervical ganglionectomy to bring about our object (*Fig 579*).

At this time Dr Ivor Davies had one of these cases under his care in a medical ward, and he readily concurred with the suggestion of Mr R D Owen and myself that if the patient consented she should be given the chance of relief which the contemplated procedure appeared to offer. Accordingly, her consent having been

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† Paterson described this change as "a thinning of the superficial epidermal layer and an apparent thickening, from infiltration, of the underlying tunica propria."

obtained, the left superior cervical ganglion was removed, and eleven days later the right one also. Figs 580-584 show the ganglia and their microscopical appearances, and the patient with a double Horner's syndrome (enophthalmos and myosis)

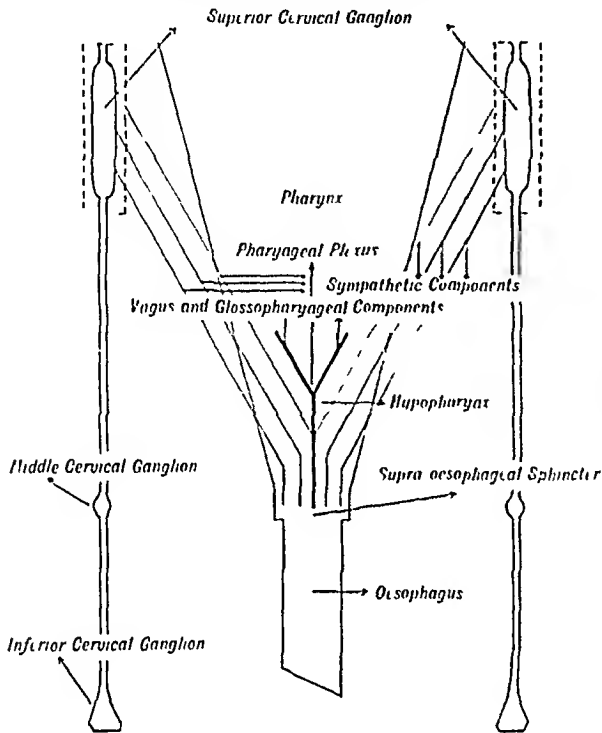


FIG 579 — Diagrammatic representation of the sympathetic innervation of the pharyngeal plexus, showing how bilateral superior cervical ganglionectomy destroys the sympathetic innervation

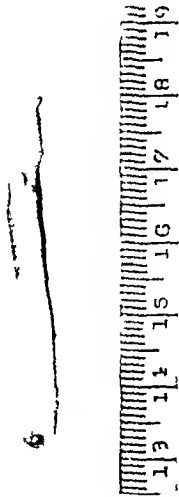


FIG 580 —Right superior cervical ganglion

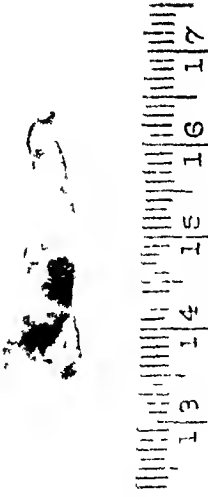


FIG 581 —Left superior cervical ganglion

consequent upon destroying the sympathetic fibres to the orbit. The Horner's syndrome does not inconvenience her in any way, in fact, vision is better through the small pupil, nor, since it is bilateral, does it detract from her appearance. She

is able to swallow food of any kind, and expresses herself very satisfied with the operation

A brief account of the case is as follows —

A married woman, aged 59, the mother of three healthy children, complained of difficulty in swallowing of two and a half years' duration and of attacks of slight breathlessness. In May, 1932, she first had difficulty in swallowing solid food, but had noticed that if she drank large quantities of water with her food, she could to a certain extent overcome the difficulty. The food appeared to stick at the suprasternal notch or rather above it, and there was an occasional burning sensation when attempting to swallow

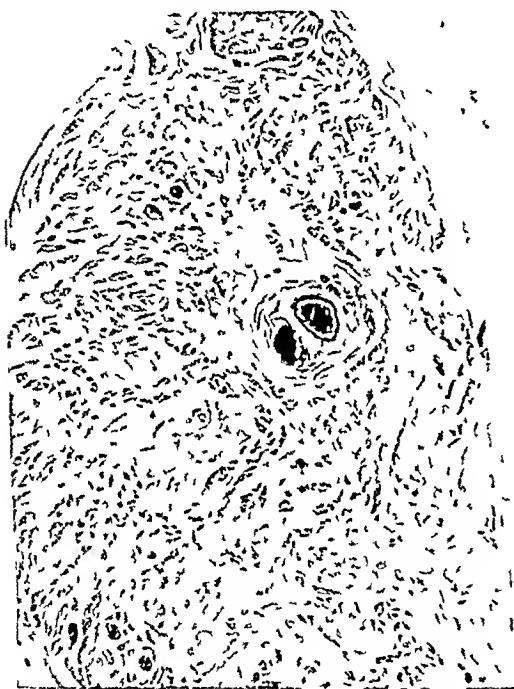


FIG 582—Histological appearance of right superior cervical ganglion

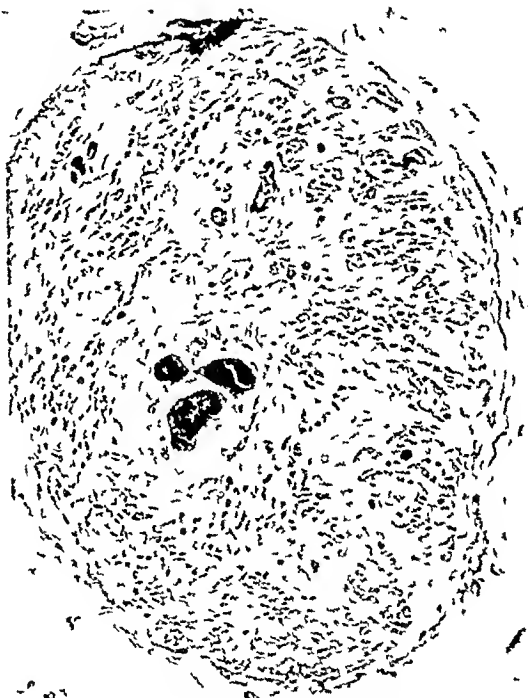


FIG 583—Histological appearance of left superior cervical ganglion

On Sept 5 she was examined by direct œsophagoscopy by Mr R D Owen, who found post-cricoid constriction and had difficulty in passing the tube behind the cricoid—the mucosa was cracked. Following instrumental dilatation she had some relief.

On Dec 30 œsophagoscopy was again performed, and the tube passed easily. The condition then relapsed, and on Oct 14, 1934, she was admitted to the Cardiff Royal Infirmary under the care of Dr Ivor Davies. Œsophagoscopy now showed slight post-cricoid hyperplasia but no evidence of new growth, the mucosa was more resilient than on previous occasions, there was less anæmia, and no cracking took place when the œsophagus was entered. A test-meal on March 1 showed achlorhydria.

On Sept 7, 1932, her erythrocytes were 3,760,000 and the hæmoglobin 65 per cent, on Sept 27, the figures were 3,040,000 and 50 per cent, on March 27, 1934,

4,240,000 and 45 per cent, on Oct 10, 4,529,000 and 54 per cent, on Oct 16, 5,808,000 and 64 per cent, and on Nov 3, 5,420,000 and 65 per cent

Examination showed a rather spare woman with a sallow complexion and a smooth tongue

OPERATION—On Nov 22, 1934, the left superior cervical ganglion was removed through a curved incision extending from behind the mastoid process downwards and forwards across the anterior part of the sternomastoid, which muscle was

retracted laterally and the carotid sheath exposed. This was opened and the vagus recognized and retracted. The ganglion was identified lying behind the sheath on the rectus capitis anticus major and longus colli muscles. The superior cardiac nerve was noted as the largest branch of the ganglion and was divided. The branches to the pharyngeal plexus were divided and the ganglion removed (see Fig 581), with its connecting sympathetic cord below its lower pole. The deep fascia and platysma were closed with catgut, and the skin with clips.

A similar operation was performed on Dec 3, 1934, on the right side, and the ganglion removed (See Fig 580)

Fig 584 shows the condition of the patient with bilateral Horner's syndrome ten days after the second operation was performed.

I have sought early publication because these cases, though not very common, are from time to time met with in most laryngological or medical

out-patient departments, and as the procedure described appears to be a logical way of dealing with them, it would seem desirable to give these unfortunate sufferers such chance as it offers. Apart from immediate relief it may obviate the onset of carcinoma. It remains for time to show whether relapses will occur, and I shall welcome the reports of others who may accept the line of reasoning submitted and be prepared to give this procedure fair trial.



FIG 584—Patient ten days after second operation
Bilateral Horner's syndrome apparent

BIBLIOGRAPHY

- ¹ PATERSON, D. R., *Four of Laryngol*, 1919, xxiv, 289
- ² OWEN, R. D., Personal communication, 1934

ON OSTEO-ARTHRITIS IN THE DORSAL INTERVERTEBRAL JOINTS A STUDY IN MORBID ANATOMY

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INTRODUCTION

THE material on which the following observations have been made consists of nothing but dried macerated bones derived from vertebral columns assembled for anatomical or anthropological research. It is greatly to be regretted that no clinical notes of any sort are available.

The purpose of this paper is to describe the occurrence of osteo-arthritis in the synovial joints of the human vertebral column, and to offer speculations on the circumstances which cause certain regions of the vertebral column to be particularly prone to the development of this disease and others to be less so.

From the outset it will be well to state plainly that the condition commonly called 'osteo-arthritis of the spine' is not the subject of discussion in this paper. The term 'osteo-arthritis of the spine' is, I think, never used to convey a meaning which is comparable with that conveyed by 'osteo-arthritis' of, say, the knee or shoulder. In osteo-arthritis of the great movable weight-bearing joints of the body no single character is so constant as the presence of osteophytes, which, at least in their early stages, grow from the edges of the areas of contact and weight bearing. In the vertebral column of man no bony disease is more common than that marked by the growth of osteophytes from the edges of adjacent vertebral bodies towards each other. By analogy, as I assume, and in the absence of precise information as to morbid anatomy, growth of osteophytes has seemed to justify the use of the name 'osteo-arthritis of the spine' for this disease.

The joint between a pair of vertebral bodies is formed by layers of fibrous tissue built upon the flat cartilage-covered surfaces of the vertebral bodies, and containing neither cavity nor synovial membrane, but a mass of semi-fluid mucin-like matter under pressure. The synovial joints of the body generally present a contrast in that each has all the following features—a cavity, cartilage-covered contact surfaces, and a vascular synovial membrane, all enclosed in a fibrous capsule.

It is in movable, synovial, or diarthroidal, joints that osteo-arthritis has been studied by a host of observers from the clinical, the pathological, and the experimental points of view. There can be no doubt that osteo-arthritis as ordinarily regarded is exclusively a disease of synovial joints.

Each unit of the vertebral column articulates with each of its neighbours by its dorsal arch and by its body. The joints between the vertebral bodies are of the sort briefly described above and ordinarily called the 'intervertebral discs' or 'fibrocartilages'. Each dorsal arch sends upwards a pair of superior articular

processes, and downwards a pair of inferior articular processes. By contact of the superior articular processes with the inferior articular processes of the arch of the vertebra placed next headward, and vice versa, is built up a chain of joints between the dorsal arches of the vertebræ. These are the dorsal intervertebral joints, and are synovial in character. It is osteo-arthritis of these dorsal intervertebral joints which is the subject matter of this paper.

Distribution in the vertebral column of osteo-arthritis of the dorsal intervertebral joints is not stated in any printed work to which I have access, indeed, in only two places have I met any reference to osteo-arthritis of these joints at all. The joints between the vertebral bodies are far larger than those between the dorsal arches, and are far more readily displayed by X-rays. This may be the reason why disease in the dorsal intervertebral joints is commonly overlooked and such clinical significance as disease in this position may have is not appreciated. Other synovial joints in the vertebral column are those made by the ribs, the costo-central and the costo-transverse, the occipito-atloid, and the anterior atlanto-axoid joints.

Robert Adams, of Dublin, is one of the earliest modern writers on osteo-arthritis. His book, *A Treatise on Chronic Rheumatic Arthritis* is entirely devoted to a study of osteo-arthritis—to use the appropriate modern term—in most of the important synovial joints of the body. The author gives an account of osteo-arthritis in the dorsal intervertebral joints and, curiously, in the anterior atlanto-axoid joint¹, but he entirely disregards the common osteophytic disease of the vertebral bodies to which the name 'osteo-arthritis of the spine' is so frequently applied. Arbuthnot Lane² records disease in these joints and also in the anterior atlanto-axoid joint in his account of the changes in the vertebral column which accompany heavy labour.

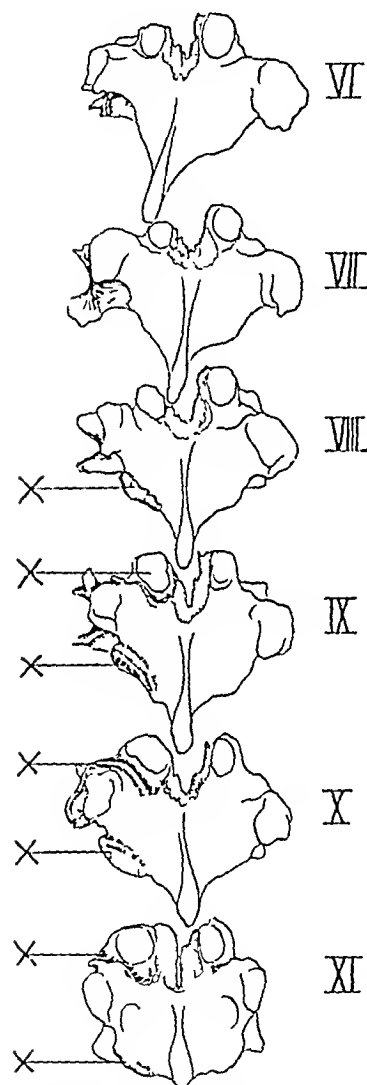


FIG 585.—A posterior view of the dorsal arches of six thoracic vertebræ set out in descending series. X indicates the presence of osteo arthritis in the dorsal intervertebral joint. Note that this change affects the dorsal intervertebral joints on the left side only. These vertebræ were found in the hollow of the scoliotic column whose concavity lies towards the left in this region, as is shown in Fig 592.

THE CAUSES OF OSTEO-ARTHRITIS

That mechanical forces have a large share in the production of this disease will probably be universally admitted, and in the vertebral column

this can be readily shown by the details of a common occurrence.

The dorsal intervertebral joints in the cavity or pressure zone of a scoliotic spine are likely to acquire the disease. Fig 585 shows osteo-arthritic changes in a series of joints, all on the left side, derived from the concavity of a scoliotic spine.

(the same as is shown in *Fig 592*) When disease grossly disturbs the normal balance of the vertebral column an almost invariable associated finding is osteo-arthritis in the dorsal joints above and below the district of disturbance

Fig 586 shows osteo-arthritis in the dorsal intervertebral joints above and below the region of gross disturbance caused by collapse of a vertebral body The link of association must be mechanical strain Among the causes of osteo-arthritis Timbrell Fisher³ notes "a localized increase of articular stress of an occupational nature or caused by mal-united fracture, disease of the limb bones with consequent deformity, or ankylosis of an adjacent joint" The principle underlying this generalization is contained in the observations I have made above In this connection the strongly expressed view of Arbuthnot Lane⁴ is worth recalling "There is no such disease as rheumatoid arthritis, but the conditions which are regarded as constituting that disease are identical with the changes produced in the osseous system by pressure" When it is noted that the 'rheumatoid arthritis' of this passage is

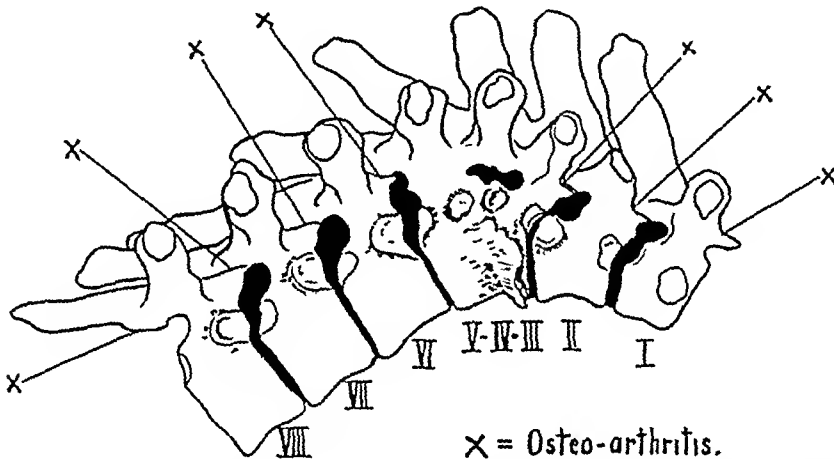


FIG 586—Part of a thoracic column viewed from the right side The 3rd, 4th, and 5th vertebrae have coalesced in one mass following collapse of the 4th vertebra as a result of disease, possibly tuberculous Angulation of the dorsal column has followed, and osteo-arthritis affects the dorsal intervertebral joints above and below the site of disturbance

the 'osteo-arthritis' of modern use, a comparison with a recent definition is interesting Osteo-arthritis is defined by Fisher⁵ as "a series of physiological and pathological changes occurring in a joint subjected to prolonged or oft-repeated injury, either mechanical or toxic, but of a moderate degree of intensity"

Osteo-arthritis might not unfairly be held to represent an effect of wear and tear on a joint There seems no doubt that central degeneration of articular cartilage is the starting-point of the chain of events that has osteo-arthritis at the other Under surgical conditions Timbrell Fisher erased the central part of one articular cartilage of a joint in a living animal The results include degeneration of a corresponding area of the opposing cartilage and the growth of peri-articular osteophytes It should follow that growth of chondrosteophytes from the two articular surfaces must progress equally, because failure of cartilage contact in any place would lead to degeneration opposite to it, certainly I have found that osteo-arthritis is equally marked in both the bones that enter into a joint

It might be surmised that each synovial joint has its normal or optimum

provision of cartilage As cartilage is lost centrally by disease, injury, or wear, the remainder is supplemented by peripheral growth in an attempt to restore the normal allowance It is growth of cartilage with its scaffolding of bone that constitutes the chondrosteophyte characteristic of the disease, and this takes place at the most vascular region, the periphery

It is not believed by modern observers that mechanical factors are the only ones that enter into the etiology of osteo-arthritis Timbrell Fisher, no less than other writers on this topic from the clinical aspect, insists on the need for elimination of any source of toxic absorption in the treatment of a patient The suggestion is that microbes or toxins may render the cartilages or the bones more open to the influence of pressure

It is not intended in this place to attempt to give even a brief review of the evidence that the products of microbic activity, disordered metabolism, and internal secretion may each have their influence It will suffice to remark that osteo-arthritis has been noted in a hæmophilic child aged 13 years,⁶ and also in a child aged 15, suffering from premature and imperfect general development—progeria⁷ Garrod⁸ has remarked on the "peculiar liability of persons with alcaptonuria and ochronosis to develop osteo-arthritic lesions in later life"

The mechanical factor is evident and denied by none For the present purpose this factor is the only one that my material allows me to consider

I can only presume that pressure between joint surfaces over an extended period is a circumstance likely to result in osteo-arthritis In the vertebral column, whose function is to bear weight in the erect attitude, this amounts to dorsiflexion between the vertebral units In dorsiflexion the articular surfaces of the joints borne on the dorsal arches are evidently brought into contact, and in ventriflexion these joint surfaces are relieved of direct pressure

In the following pages I shall present to the reader observations of facts relating to the occurrence of osteo-arthritis, and shall offer speculations on the means by which mechanical pressure might exert an influence in the production of osteo-arthritis in the dorsal intervertebral joints

ANATOMY OF OSTEO-ARTHRITIS AS SEEN IN THE DORSAL INTERVERTEBRAL JOINTS

In my survey, so long as peripheral osteophytes could be recognized, the joint in question was regarded as the subject of osteo-arthritis In advanced states the diseased joint surfaces may be grooved, eburnated, and greatly expanded by the formation of new bone

In a large number of vertebræ will be found all of the following bones of which the articular contact surfaces are clearly marked and the evidence of osteo-arthritis is limited to circumferential osteophytes, bones whose contact surfaces having no vestige of normal structure present irregular surfaces, here polished, there porous, as if treated first with a rasp and then with a burnisher, but still defined by heaped osteophytes around, and every stage between these two extremes

In *Fig 587* is shown a photograph, and in *Fig 588* an enlarged scale drawing, of the surface of a flat intervertebral joint moderately advanced in osteo-arthritis Three areas which are approximately circular and concentric can be recognized —

Area A—A central area which corresponds to the cartilage-covered area of a normal joint. This area encloses bone of fine texture and is neither porous nor polished.

Area B—An intermediate ring of porous bone marked by holes which vary considerably in size and depth. Many of these holes would admit a pin, some are shallow pits, and others penetrate the bone deeply.

Area C—A third zone consists of a rampart of irregular osteophytes often

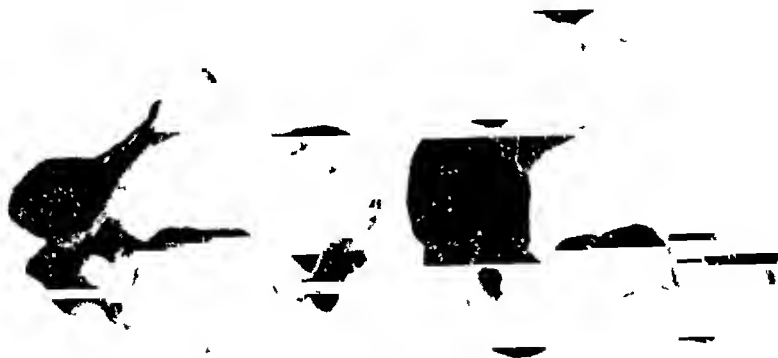


FIG 587—The photograph on the right is of the lower aspect of a specimen of the 2nd cervical vertebra. On the left is shown the upper aspect of the 3rd cervical vertebra with which it makes contact. Note the osteoarthritic joint on the left side of the vertebra, which is portrayed in more detail in a line enlargement in Fig 588. The spreading osteophytes from the diseased joint, it may be observed, go far to obstruct the groove for exit of the third cervical nerve.

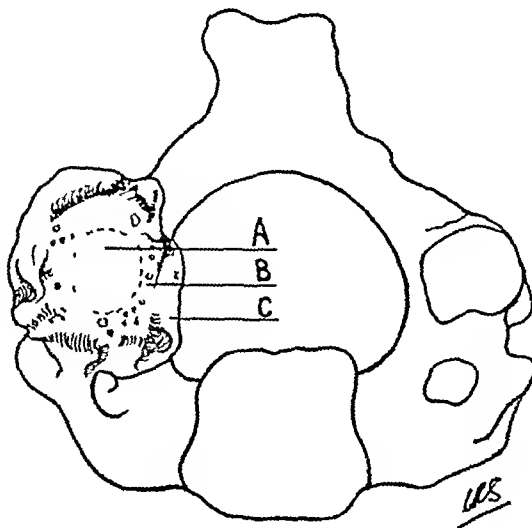


FIG 588—Enlarged line drawing of the upper surface of the second left intervertebral joint shown in Fig 587. Note the three areas shown: A, Primary contact area; B, Zone of porous bone; C, Rampart.

discontinuous but encircling the intermediate ring and conspicuous chiefly by their irregularity. These osteophytes are seemingly not of porous bone, but are often highly polished.

By the characters of the contact surfaces it is possible to classify osteo-arthritis

of the small and simple intervertebral joints in three stages, which I conceive to represent consecutive steps in the development of osteo-arthritis —

Stage I—The primary contact *Area A* bears osteophytes at its margin. Only *Areas A* and *C* are present, and it must be supposed that expansion of the contact area has only just begun.

Stage II—All three areas, *A*, *B*, and *C*, are present. Expansion has gone a stage further, and the area of secondary expansion, *B*, is exposed as the rampart recedes. The fact that this *Area B* is porous and sometimes shows blood staining in recently prepared bones, suggests that it is a vascular region. It is probable that the spaces of the vascular bone-marrow are being exposed.

Stage III—The limits of *Area A* are no longer to be seen. The contact area may be all of porous bone or it may be grooved and eburnated. The rampart *C* may be very conspicuous, or it may, on the contrary, be broken down and worn flush. Eburnation may be found in any part of the articular area at any stage.

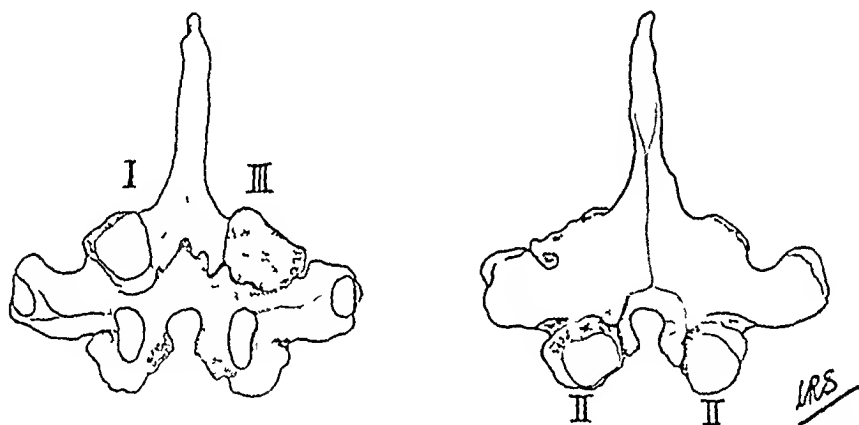


FIG. 589.—Drawings of the dorsal arch of a specimen of the 7th thoracic vertebra. The dorsal arch is viewed from the dorsal aspect in the right-hand drawing and from the ventral aspect in the left-hand. By each of the four articular facets is placed a Roman numeral which indicates the stage of development of osteo-arthritis.

Examples of these three stages are shown in the drawings in *Fig. 589*. Timbrell Fisher, under circumstances which are not available to me, has observed osteo-arthritis in all its aspects, and has based on his pathological findings a classification⁹ which I set out for comparison with mine. Fisher's classification, being based on post-mortem observations of very wide range, is far more complete than mine, which is based on a material restricted to dried bones.

The persistence of the normal contact area of a joint is very striking. Though the surface of actual contact is doubled, still this area may be recognizable. An explanation is perhaps to be found in Fisher's¹⁰ observation that "the layer of bone underneath the articular cartilage can be seen to be thickened before the cartilage covering it has entirely disappeared."

It may be remarked that the small dorsal intervertebral joints are peculiarly suitable for observations of the sort I have made. The articular surfaces are small but even in outline, most of them are flat, and the rest only slightly curved.

TIMBRELL FISHER'S CLASSIFICATION	CLASSIFICATION BASED ON PATHOLOGICAL ANATOMY OF BONE (<i>supra</i>)
<i>Class I</i> Fibrillation and earlier changes in the central area of the articular cartilage	—
<i>Class II</i> Fibrillation more marked, commencing lifting of articular margins, with hyperplasia of the synovial membrane	<i>Stage I</i>
<i>Class III</i> Changes more marked, commencing exposure of sub-articular bone, and more generalized changes in synovial membrane. Deformity from chondro-osteophytic formations	<i>Stage II</i>
<i>Class IV</i> Bone extensively exposed, often eburnated and grooved. Destruction of intra-articular ligaments. Synovial fringes usually fibrosed or atrophic. Deformity increased by shortening or sub-luxation	<i>Stage III</i>

THE DISTRIBUTION OF OSTEO-ARTHRITIS OF THE DORSAL JOINTS IN THE VERTEBRAL COLUMN

The material examined includes 126 human vertebral columns, all dried and macerated. These columns, though assembled, have not always been complete in every member. The total numbers for the several vertebrae vary from 90 to 125. In order to present the true proportional incidence of osteo-arthritis on the different parts of the vertebral column, I have made a series of simple calculations and have set out the result in a graph (*Fig. 590*) as if the total number of columns were 100. I examined all the available vertebral columns and noted the joints which displayed clear evidence of osteo-arthritis. In my notes I did not distinguish the stages of development which I have described, but merely recorded the presence of osteo-arthritis. It will be seen from *Fig. 590* that osteo-arthritis affects the dorsal intervertebral joints in three main 'outcrops', which may be defined and named as follows:

1 LUMBO-DORSAL OUTCROP—A fairly regular curve extending from the 8th thoracic vertebra to the lumbo-sacral junction.

2 DORSO-CERVICAL OUTCROP—Irrregular in form and including the joints between the 6th cervical and the 7th thoracic units. Two peaks of especially high incidence occur: (a) At the joint between the 4th and 5th thoracic vertebrae. This may be called the *upper thoracic peak*. (b) At the cervico-thoracic junction, the joint between the 7th cervical and the 1st thoracic vertebrae. This may be called the *cervico-thoracic peak*.

3 CERVICAL OUTCROP—A curve of fairly regular form between the 1st and the 7th cervical vertebrae.

Commentary on Distribution—It is necessary to inquire why osteo-arthritis has an unequal incidence on the dorsal joints of different districts of the vertebral column. In general, osteo-arthritis of the dorsal intervertebral joints assumes a higher incidence as the column is followed downwards. This suggests that weight-bearing is an important causative factor, but the presence of maximum and minimum zones shows the need for further examination of the mechanical

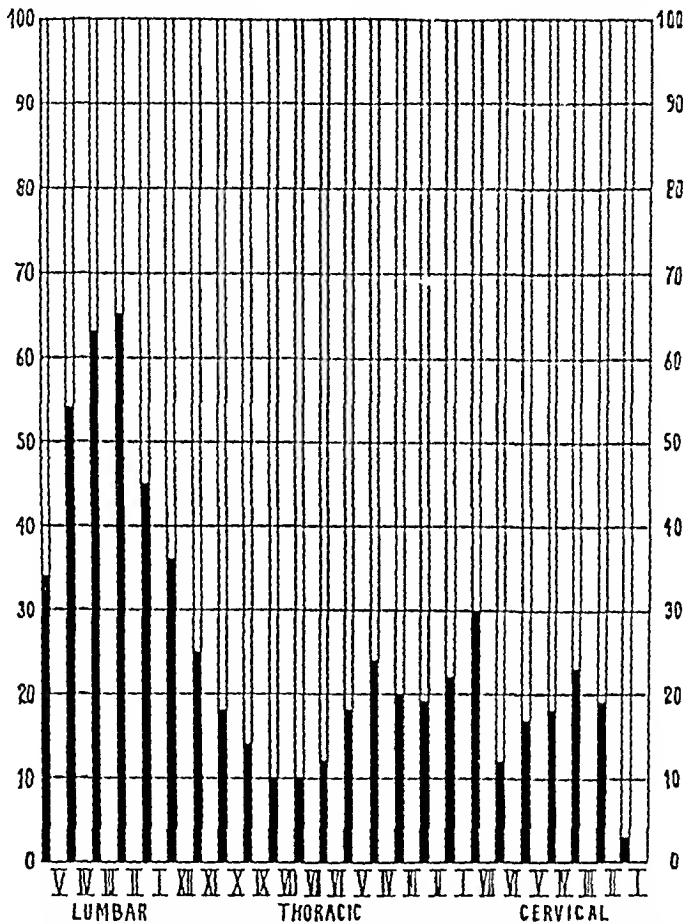


FIG. 590.—Graph showing the distribution of osteo arthritis of the dorsal intervertebral joints in the vertebral column. The abscissæ represent the vertebrae set out from left to right in ascending order. The ordinates show the proportional incidence of osteo arthritis on the various joints as if the total number of joints examined were 100. Note the three main outcrops—lumbo dorsal, dorso cervical, and cervical—which are separated by zones of minimal incidence at the joints between the 7th and 9th thoracic and at the joint between the 6th and 7th cervical vertebrae. Note the upper thoracic peak at the joint between the 4th and 5th thoracic vertebrae, and the cervico thoracic peak at the cervico thoracic junction.

circumstances under which the vertebral column carries weight. The condition under which a given joint bears a direct share in weight-bearing is that of dorsiflexion, and the presence of dorsiflexion lies in the architecture of the vertebral column. Humphry¹¹ writes on the alternating antero-posterior curves formed by the mobile units of the vertebral column as follows: "The three upper curves—the cervical, dorsal, and lumbar—are so arranged that their chords are in the same vertical line in the erect position of the body, and that vertical line corresponds with

the line of gravity of the head The centre of gravity of the cranium with its contents is placed immediately over the heads of the thigh bones, and the points of confluence of three of the intermediate curves are in the line of gravity." It follows from this statement that the units of the cervical and of the lumbar curves are dorsiflexed upon each other, lying as they do in front of the line of gravity and weight-bearing. Hence it follows that the dorsal intervertebral joints in these two curves take a direct share in bearing the weight of the column.

In the thoracic curve, however, the line of gravity lies in front of the vertebræ, which are therefore ventriflexed upon each other. Under weight-bearing the tendency is for the dorsal arches to fly apart. The dorsal intervertebral joints do not ordinarily carry weight in the same way as do those in the cervical and the lumbar curves. On the contrary, in the thoracic curve the dorsal arches are restrained from separation by the interlamina ligaments—the ligamenta subflava. In another place¹² I have shown that ossification commonly occurs in these interlamina ligaments and that this occurrence is almost confined to the thoracic region of the spine, I suggest that this effect is the result of strain.

We will consider seriatim the causes of the three outcrops of osteo-arthritis in the dorsal intervertebral joints to which reference has been made.

THE LUMBO-DORSAL OUTCROP OF OSTEO-ARTHRITIS

This outcrop of osteo-arthritis commences as a fairly even curve below the 8th thoracic vertebra, rises to a maximum at the joint between the 2nd and 3rd lumbar vertebræ, and falls away to the lumbo-sacral junction. This outcrop extends over more vertebræ in a headward direction than are ordinarily included in the lumbar curve. In height this curve of incidence exceeds any other, and therefore may be presumed to indicate the site of first development of osteo-arthritis in the dorsal intervertebral joints.

The effect of osteo-arthritis of the dorsal joints on the contour of the lumbar column is to accentuate the posterior hollow of the loin, with the result known to clinicians as 'lordosis', as is shown in Fig 591. A result is to throw back the thorax upon the lumbar spine and to bring intervertebral joints under mechanical influences

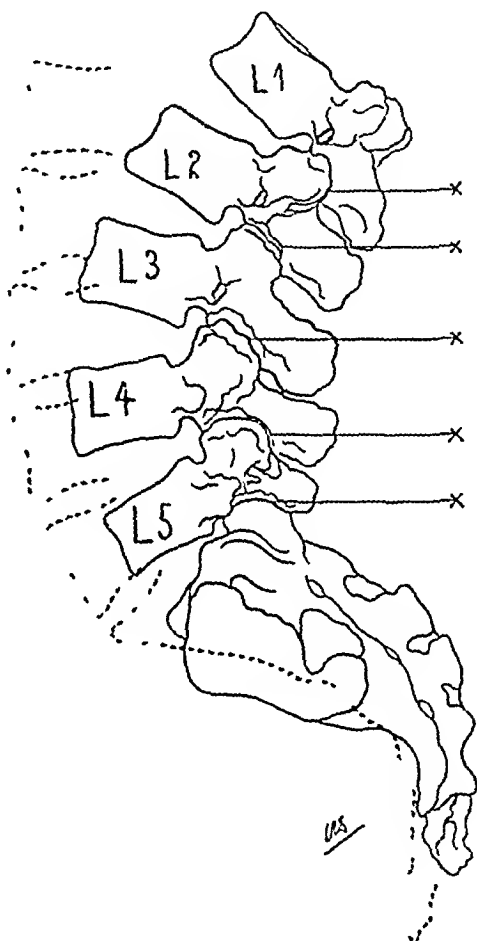


FIG 591—Tracing of the lumbar vertebræ with the sacrum reconstructed and viewed in a true lateral position. The dorsal joints indicated by X are all affected with osteo-arthritis. For the purposes of comparison the contour of the corresponding units of a normal column has been superimposed in dotted outline. This superimposed contour was traced from one of Humphry's normal specimens preserved in the Anatomy Museum at Cambridge. It is to be inferred that osteo-arthritis of the dorsal intervertebral joints leads to accentuation of the lumbar curve, i.e., to lordosis.

which properly belong to intervertebral joints of the lumbar curve only. We might call this result 'lumbarization' so far as it affects the thoracic vertebræ. I have no means of proving the surmise, but I should expect that the plumb-line from the skull now falls through a vertebra higher than the normal 12th thoracic.

This process of lumbarization is necessarily progressive once it is initiated. The lumbar curve filches more vertebræ from the thoracic curve and brings them under influences that require them to bear weight by their dorsal joints, and so to develop osteo-arthritis in their turn. Concomitant with this change at the lower end of the thorax is a forward projection of the upper thorax in order to preserve body balance. This change constitutes 'kyphosis', which brings its own immediate problems.

I suggest that the whole outcrop of osteo-arthritis on the dorsal intervertebral joints below the level of the 8th thoracic vertebra is to be interpreted as a first consequence of weight-bearing by the units of the lumbar curve through their dorsal joints and as an extension of the lumbar curve into the thoracic region with the development of lordosis.

THE DORSO-CERVICAL OUTCROP OF OSTEO-ARTHRITIS

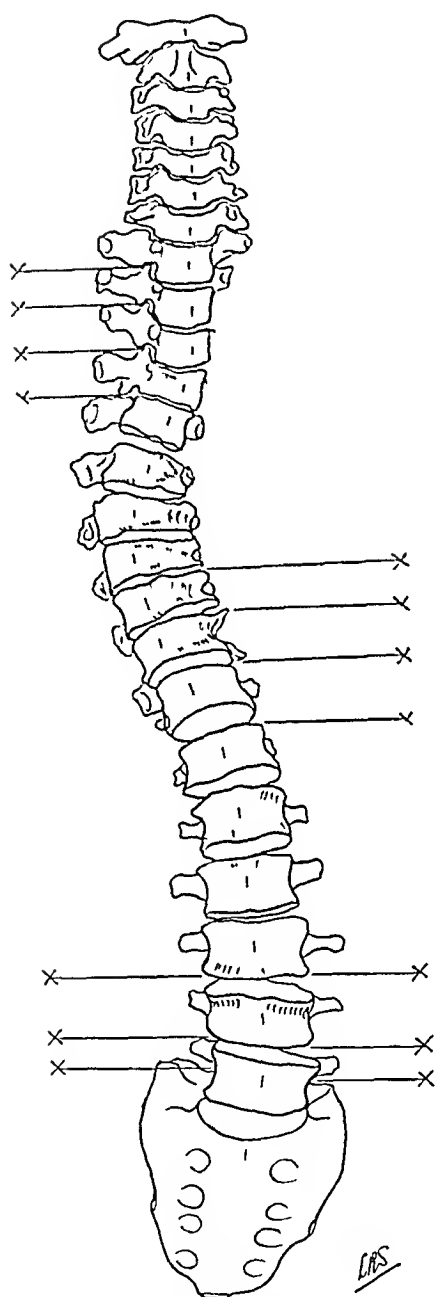
The district covered by this outcrop, between the 6th cervical and the 7th thoracic units, should, according to the principles enunciated, be relatively free from osteo-arthritis of the dorsal intervertebral joints. The vertebræ of this region are ordinarily ventrified on each other, and vertical strains are met by ligaments rather than by joint surfaces.

An irregular zone of incidence includes the joint between the 7th cervical and the 1st thoracic vertebræ. This high *cervico-thoracic peak*, close to a point of low incidence at the joint next above it, being indeed the maximum point of the whole dorso-cervical outcrop, makes a strong contrast with the even cervical outcrop. The next highest incidence of osteo-arthritis in the dorso-cervical outcrop is at the joint between the 4th and 5th vertebræ—the *upper thoracic peak*.

It will be most convenient to consider first a possible cause of the whole dorso-cervical outcrop, and then the special cause for the *upper thoracic* and the *cervico-thoracic peaks*. The presence of the whole dorso-cervical outcrop is to be sought in factors that may cause dorsiflexion between the vertebræ of this region.

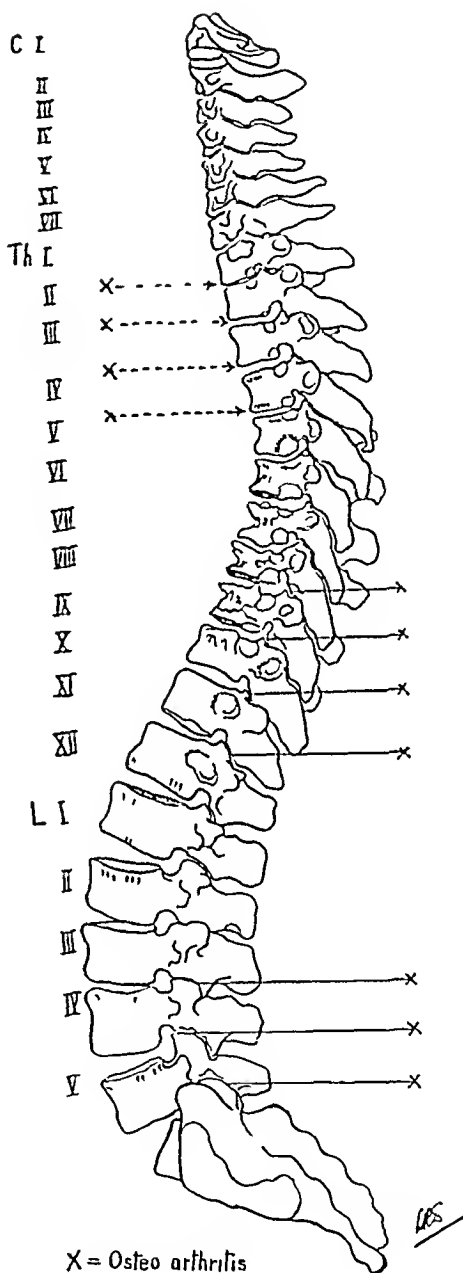
Two vertebral columns I have examined suggest that use of the upper limbs and the connection between them and the thoracic cage may account for the dorso-cervical outcrop as a whole. Much of the upper limb musculature is attached directly to the thoracic cage. The rhomboidei and the trapezius muscles connect the scapula to the thoracic spines. The scapula is strutted to the thorax by the clavicle, and many muscles besides those of the upper arm proper gain attachment to the limb girdle, such as the trapezius and the pectorals. Strong movements of the upper limbs may well imply dorsiflexion of the upper dorsal spine if accompanied by contraction of corresponding parts of the erector spinæ. I might suggest that movements such as those of rowing imply a direct strain from the upper limbs to the upper thoracic spine.

I propose to describe first a column with scoliosis of such character as to suggest the consequences of severe labour performed by the right arm and shoulder. A second column is much less deformed, but is not dissimilar to the first in its essential features. The details of osteo-arthritis of the dorsal intervertebral joints in these



X = Osteo arthritis

FIG 592 —Anterior view of a reconstructed column affected with scoliosis. X marks the position in which osteo-arthritis of the dorsal intervertebral joint is to be found. Probably severe labour performed by the right arm and shoulder accounts for the curvature of the upper dorsal spine and for osteo arthritis in the dorsal joints of that region. Osteo arthritis is also found in the dorsal joints of the compensatory lower dorsal curvature on the left side. The joint lesions of the lumbar spine are bilateral. The same column is shown in side view in Fig 593.



X = Osteo arthritis

FIG 593 —Lateral view of the same column as is shown in Fig 592. As before, X indicates osteo arthritis in the dorsal intervertebral joint. The drawing shows disease in the same positions as Fig 592, and in addition, lordosis developing in the lumbar spine.

two columns may be found to give support to the suggestion that the dorso-cervical outcrop and the use of the upper limbs are associated phenomena

COLUMN No 1—Viewed from the front (Fig 592), there appear an upper thoracic curve open to the right, a middle and lower thoracic curve with concavity open to the left, and a slight lumbar curve with concavity open to the right. Viewed from the left (Fig 593), the thoracic curve is much accentuated in its lower members, the cervical curve is somewhat straightened and continues farther than normal into the thorax, the lumbar curve is accentuated and includes the lower two thoracic vertebra. Rotation of certain vertebral units is shown by the loss of alinement of the vertical marks on the middle anterior points of the vertebral bodies in Fig 592.

In the *upper dorsal curve* osteo-arthritis has affected the joints between the first five vertebrae on the right side only. In the *lower dorsal curve* osteo-arthritis is marked only in the joints from the 8th to the 11th vertebrae on the left, i.e., on the side contralateral to the changes in the upper dorsal region. Osteo-arthritis also affects the costovertebral joints below the 5th thoracic vertebra on the left side. This is probably the result of effort to balance the trunk on the part of the muscles of the anterior abdominal wall, which extend from the lower seven ribs to the pelvis.

In the *lumbar region* osteo-arthritis occurs in the joints between the 3rd lumbar and the lumbo-sacral joint, and is bilateral. Some lordosis is to be inferred, because the vertical line seems to pass through the 10th thoracic vertebra.

This column owes its conspicuous features almost certainly to heavy labour performed by the right arm and shoulder. The contours approximate very nearly to those of the classical specimen described by Arbuthnot Lane to illustrate the effects on the skeleton of heavy labour of the sort performed by the brewer's drayman, who habitually carried heavy barrels on his right shoulder.

In the *upper thoracic region* of this column osteo-arthritic lesions of the intervertebral joints are limited to the right side. In the

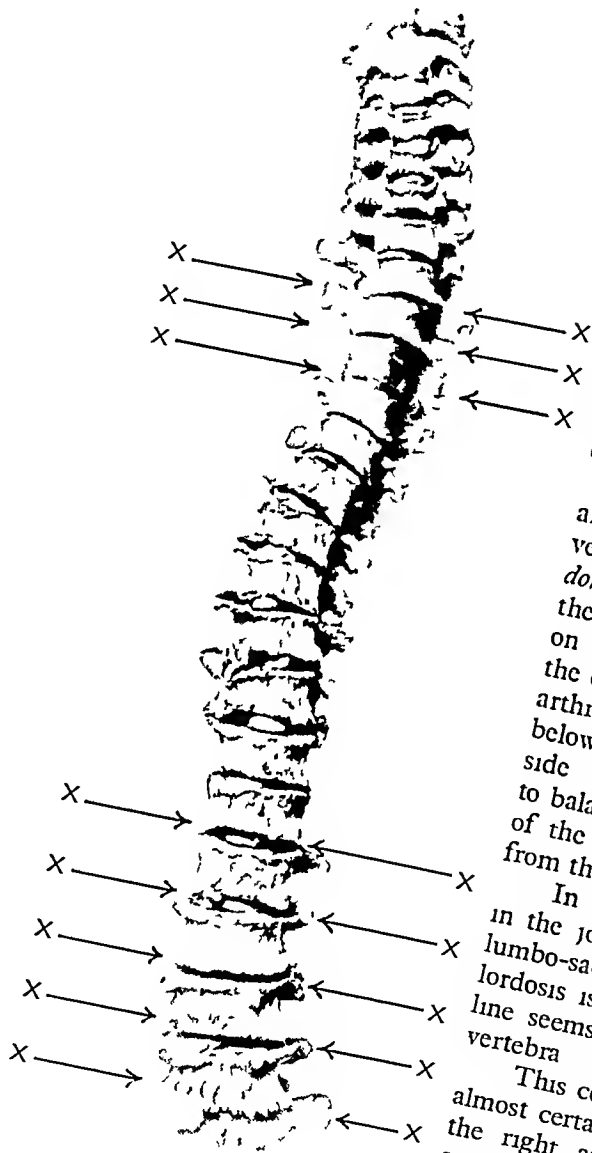


FIG 594.—A photograph of a reconstructed vertebral column from the front. Note the curvatures of the spine which are similar to those shown in Fig 592, though much less pronounced. X indicates osteo-arthritis in the dorsal intervertebral joint in the position indicated.

complete absence of any gross disease the association of strain transmitted through the right shoulder is too obvious to overlook. Left-sided lesions occur in the left compensatory curve, and bilateral lesions of pure weight-bearing in the lumbar region.

COLUMN No II—This column is shown after reconstruction from a front view in *Fig 594*. There is curvature of slight degree of the cervical and the upper dorsal spine with the concavity to the right, and of the lower dorsal and the lumbar spine in the opposite direction. Osteo-arthritis of the dorsal intervertebral joints is present in the middle of the cervical curve, between the 1st and 4th thoracic vertebræ and from the 12th thoracic vertebra to the lumbo-sacral joint. In each district the disease is bilateral.

In this column contours are similar to those in Column I, but much less pronounced. Approximately the same district of osteo-arthritis is found in the dorsal intervertebral joints of the upper thoracic region, with the difference that the osteo-arthritic changes are bilateral. I do not think that bilateral distribution invalidates the supposition that osteo-arthritis in the dorsal intervertebral joints of the upper thoracic region is associated in a causal sense with the use of the upper limbs. I should surmise that the individual from whom this second column was derived was not subject to severe asymmetrical labour comparable to that of the brewer's drayman, but was an active man with his hands and right-handed. Only few occupations might furnish examples of excessive use of the right upper limb comparable to the labour of the brewer's drayman, the ordinary activities of labour and of sport demand the use of both arms.

I have specially examined a series of skeletons to determine the side of more frequent occurrence of osteo-arthritis in the dorsal joints between the 7th cervical and the 7th thoracic vertebræ. I find that right-sided lesions constitute 54 per cent of the total, and suggest that this slight preponderance of right-sided disease is associated with the general right-handedness of man. However, the study of the details of Columns I and II suggests to me that the upper dorso-cervical outcrop of osteo-arthritis in the dorsal intervertebral joints is caused by the use of the upper limbs. Such is the provisional explanation I offer for the occurrence.

Frazer¹³ remarks that "a slight lateral curve is sometimes seen in the lines of the bodies, concave to the left in the dorsal region and probably due to the use of the right arm, with some compensatory deviation above and below." The columns we have been examining therefore show an exaggerated state of the normal in the matter of their contours.

The Upper Thoracic Peak of the Dorso-cervical Outcrop—The presumption is that the peak between the 4th and 5th thoracic vertebræ indicates pressure by dorsiflexion in the dorsal intervertebral joints of that district. In this connection we must remember the special function which is attached to the thorax, namely, that of respiration. Halls Dally¹⁴ showed by X-ray examination that straightening of the whole thoracic spine is a normal accompaniment of the inspiratory phase of respiration. The spinous processes are approximated and closely overlap, as Halls Dally suggests, in order to form a natural protection against hyper-extension. The vertebral bodies show divergence anteriorly as the intervertebral discs open out and the anterior common ligament becomes taut, and reverse movements accompany the expiratory phase. It must surely follow that the dorsal joints of the thoracic vertebræ become places of pressure with each act of inspiration,

in this case it cannot be a matter for wonder if these joints show the effects of wear and are prone to the development of osteo-arthritis. The normal wear at the part of the thoracic curve that corresponds to the upper thoracic peak might very probably become intensified under certain circumstances.

We have seen how osteo-arthritis in the dorsal joints of the lumbar spine is associated with lordosis, and we have surmised a concomitant kyphosis with reduction of the functional length of the thorax. It is possible that in order to compensate for this reduction of the thoracic spine and of the respiratory tide, the normal excursion of the thoracic spine in dorsiflexion may be increased. Such an event would be expected to throw additional stress upon the dorsal joints.

The concurrence of two circumstances, the use of the upper limb and the respiratory movements, both of which accompany dorsiflexion, may account for the high incidence of osteo-arthritis in the dorsal intervertebral joints at the *upper cervical peak*.

The Cervico-thoracic Peak of the Dorso-cervical Outcrop—I suggest that the reason why a peculiar strain of dorsiflexion falls on the joints between the 7th cervical and the 1st thoracic vertebræ may lie in the adjustments forced upon the column in order to maintain the poise of the head in the face of alteration of curvature of the spine elsewhere. In the evolution of man perhaps there has been no factor of more constant significance than that of keeping the head erect in order to maintain forward vision. When it is considered what a handicap is placed upon a man if his trunk is bowed forwards, or what a strain is endured if he assume for only the short period of an experiment the attitude of a quadruped, the force of the urge to keep the head upright and poised will be appreciated. No matter how much the trunk is bent, every effort is made to keep the head upright. When the thorax is bowed forwards in kyphosis, then dorsal angulation between neck and thorax must be maintained at whatever level it is necessary to secure the poise of the head.

This surmise may be confirmed from the examination of any series of deformed columns in our museums. In the most deformed columns the neck is commonly found sharply dorsiflexed on the thorax. I suggest that the cervico-thoracic peak of osteo-arthritis may be interpreted in these terms. It is an indication of a compensatory change as the neck forms a new angle with the thorax, and might be described as a result of kyphosis.

THE CERVICAL OUTCROP OF OSTEO-ARTHRITIS

This outcrop of osteo-arthritis extends from the first to the sixth cervical joints, with its maximum at the joint between the 3rd and 4th vertebræ.

The dorsal joints of the vertebræ of the cervical curve carry weight as do those of the lumbar curve, because the vertebral bodies lie in front of the line of gravity. Therefore these joints are specially prone to the development of osteo-arthritis, and as might be expected the point of greatest incidence is near the middle of the curve. Humphry states that the fore part of the 4th vertebra forms the zenith of the curve.

It could be imagined that the uppermost thoracic vertebræ might become functionally absorbed into the cervical curve if lordosis demands a forward projection of the thorax in order to maintain body balance. If this be so, as disease of the dorsal joints spreads the thorax must become functionally shortened, certainly

at the lower end and perhaps at the upper end also. It is remarkable if the adjustments of the trunk to the changing contours of the spine are affected at the expense of the thorax. There is room for research into the change in vital capacity that may accompany lordosis and kyphosis.

However this may be, it is difficult to explain the very high incidence of osteo-arthritis at the cervico-thoracic junction as an extension of the cervical curve. A special local function explaining that occurrence has already been discussed.

OSTEO-ARTHRITIS OF THE ANTERIOR ATLANTO-AXOID JOINT

Out of all the columns showing osteo-arthritis I examined, 32.2 per cent of specimens of the atlas and axis showed disease in this joint. Comparison of this figure with the graph in *Fig 590* will show that osteo-arthritis is commoner in the anterior atlanto-axoid joint than in any of the dorsal intervertebral joints, except in those of the lumbar region.

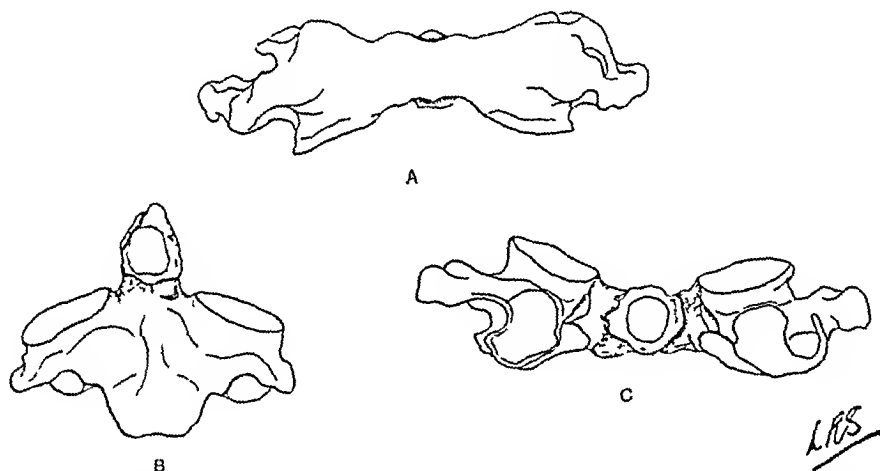


FIG 595.—Drawings showing A, The anterior arch of the atlas from the front with projecting osteophytes in the middle, B, The front of the axis with odontoid process, and C, The anterior arch of the axis from the back after the posterior arch has been removed. B and C show the two contact surfaces of the anterior atlanto-axoid joint, note peripheral osteophytes indicating early osteo-arthritis (Stage I).

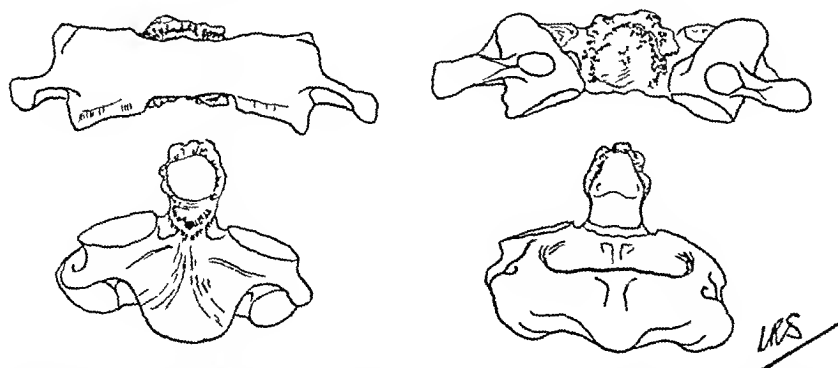


FIG 596.—Drawings of a specimen of the atlas and of the axis vertebra showing osteo-arthritis of the anterior atlanto-axoid joint of a more advanced degree (Stage III). The two upper drawings show the anterior arch of the atlas from the front (left) and the back (right). The two lower drawings show the axis and the odontoid process from the front (left) and the back (right). Cross-hatching in the upper right drawing indicates eburnation.

The contact surfaces of this joint are the front of the odontoid process of the axis and the back of the anterior arch of the atlas, and the movement in this joint is universally conceded to be rotatory. It is remarkable that Adams, to whom reference has already been made, gives to disease of this joint a very large share of his account of osteo-arthritis of the small synovial joints of the vertebral column. He wrote¹⁵ "the atlas and the dentata have far more free movements on each other than any of the other vertebræ enjoy, a circumstance that may account for the observation that we are accustomed to see far more numerous specimens of the effects of chronic rheumatic arthritis in these vertebræ than in all the others of the spine taken collectively." My findings do not confirm this final statement, but certainly osteo-arthritis of the anterior atlanto-axoid joint is very common.

Drawings showing disease in this joint will be found in *Figs* 595 and 596. Arbuthnot Lane notes osteo-arthritis ('rheumatoid arthritis') of this joint in his account of labour changes and the effects of weight-carrying. It is described in connection with the occupation of the coal-heaver, who carries weight on the back supported by the head and neck through the medium of a long-flapped cap.

Osteo-arthritis of the anterior atlanto-axoid joint might well have a clinical interest. In this position it might very well be the cause of occipital headache and of stiff neck if Hilton's¹⁶ well-known generalization is accepted. "The same trunks of nerves, whose branches supply the groups of muscles moving a joint, furnish also a distribution of nerves to the skin over the insertions of the muscles, and the interior of the joint receives its nerves from the same source." Probably the anterior atlanto-axoid joint receives its nerve-supply from the first cervical nerve, which lies very close to the joint and supplies the short rotator and extensor muscles of the suboccipital space and the semispinalis capitis of the nuchal region also. The skin does not receive a direct supply from the first cervical nerve, but by reason of a communication with the second cervical, the first cervical nerve takes a share in the supply of an area of skin to which the great occipital nerve is distributed. Therefore, an area of skin extending from the vertex to the nuchal crest and from mastoid process to mastoid process might be the seat of sensation referred from the anterior atlanto-axoid joint by the first cervical nerve.

SUMMARY AND CONCLUSIONS

This paper is devoted to the consideration of osteo-arthritis of the small synovial joints of the vertebral column. This condition is to be distinguished from 'osteo-arthritis of the spine'. In a description of the pathological anatomy of osteo-arthritis three stages of development are described. In the first the disease is indicated only by a fringe of osteophytes around the normal contact area, in the second by a zone of porous bone which separates the original contact area from a peripheral fringe of osteophytes, in the third stage all traces of the original contact area are lost and the surface may be grooved, polished, and greatly deformed.

The distribution of osteo-arthritis in the dorsal intervertebral joints, which has not previously been recorded, is set out in graphic form, and certain zones of special incidence are observed.

I Lumbo-dorsal Outcrop—This is interpreted as the result of weight-bearing in the joints of the dorsiflexed lumbar column, and the absorption of the lower thoracic vertebræ into the lumbar curve as lordosis is established.

2 **Dorso-cervical Outcrop**—It is suggested that in the main this outcrop is the result of the use of the upper limbs, by movements of dorsiflexion transferred from the limbs to the thoracic skeleton and by associated action of the erector spinæ muscle. This outcrop presents two peaks of higher incidence —

a At the joint between the 4th and 5th thoracic vertebræ is found the *upper thoracic peak*. It is suggested that this peak is due to dorsiflexion which is a normal feature of inspiration accentuated with the onset of lordosis and kyphosis.

b The cervico-dorsal junction is marked by a *cervico-dorsal peak*. It is suggested that the explanation lies in the powerful urge to keep the head upright in spite of kyphotic changes in the thorax. Columns greatly deformed with kyphosis often show that the head and neck are borne upright in spite of great postural difficulties.

3 **Cervical Outcrop**—This is probably due to weight bearing in the joints of the already dorsiflexed cervical vertebræ.

Some space is given to a description of osteo-arthritis in the anterior atlanto-axoid joint. Disease in this position was found in about one-third of columns which exhibit osteo-arthritis elsewhere in the dorsal intervertebral joints.

I have pleasure in making grateful acknowledgements to those who have assisted me by providing facilities for observation, or by other means. Professor R. A. Dart, of the University of the Witwatersrand, allowed me full access to the collection of skeletons in the Anatomical Museum. It was here that the first observations were made. My investigations were continued at Cambridge, where Professor J. T. Wilson and Dr W. L. H. Duckworth gave me full opportunity of examining the skeletons in the Anatomy Department. Sir Walter Langdon-Brown, Regius Professor of Physic at Cambridge, and the M.D. Committee permit me to publish these observations, which formed part of a thesis presented for the degree of M.D. in June, 1933.

REFERENCES

- ¹ ADAMS, ROBERT, *A Treatise on Chronic Rheumatic Arthritis*, 1857, 283. London.
- ² LANE, W. A., "Some Points in the Physiology and Pathology of the Changes produced by Pressure in the Bony Skeleton of the Trunk and Shoulder Girdle", *Guy's Hosp. Rep.*, 1886, xliii, 321.
- ³ FISHER, TIMBRELL, *Chronic Non-tuberculous Arthritis*, 1929, 118.
- ⁴ LANE, W. A., "The Causation and Pathology of the So-called Disease, Rheumatoid Arthritis, and of Senile Changes", *Trans. Pathol. Soc. Lond.*, 1886, xxxvi, 387.
- ⁵ FISHER, TIMBRELL, "A Contribution to the Pathology and Etiology of Osteo-arthritis", *Brit. Jour. Surg.*, 1922, x, 73.
- ⁶ LECG, WICKHAM, "A Second Case of Hæmophilia with Examination of the Tissues and Joints", *Trans. Pathol. Soc. Lond.*, 1885, xxxvi, 412.
- ⁷ HASTINGS, GIFFORD, "On a Condition of Mixed Premature and Immature Development", *Med.-Chir. Trans.*, 1897, lxxv, 17.
- ⁸ GARROD, SIR ARCHIBALD E., *Proc. Roy. Soc. Med.*, 1923, xvii, 2.
- ⁹ FISHER, TIMBRELL, *Loc. cit.* (3).
- ¹⁰ FISHER, TIMBRELL, *Loc. cit.* (3), 50.
- ¹¹ HUMPHRY, G., *The Human Skeleton*, 1858, 147.
- ¹² SHORE, L. R., "A Report on the Nature of Certain Bony Spurs arising from the Dorsal Arches of the Thoracic Vertebræ", *Jour. of Anat.*, 1931, lxxv, 379.
- ¹³ FRAZER, J. E., *The Anatomy of the Human Skeleton*, 1914, 14.
- ¹⁴ DALLY, HALLS, 1908, "The Movements of Respiration", *Jour. of Anat. and Physiol.*, 1908, xliii, 98.
- ¹⁵ ADAMS, ROBERT, *Loc. cit.*, 287.
- ¹⁶ HILTON, JOHN, *Rest and Pain*, 1880, 3rd ed., 168.

POLYSPONDYLITIS MARGINALIS OSTEOPHYTICA

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INTRODUCTION

A COMMON disease can be recognized in *Fig 597*, which is the photograph of a vertebral column reconstructed from its constituent bones. The drawing and the radiogram reproduced in *Fig 598* will perhaps also recall the same familiar condition. So far as I can find, this disease, though probably the commonest to which the vertebral column of man is subject, has no precise name. 'Spondylitis alone—or perhaps with the epithet 'hypertrophic'—'spondylitis deformans', 'spondylosis', and 'osteo-arthritis of the spine' probably include most of the synonyms in common use.

No definite accounts are to be found in standard works on pathology, medicine, or surgery of the anatomy of these osteophytes or of their distribution in the vertebral column. The observations on these points recorded in this paper were made upon vertebral columns which had been macerated, dried, and rendered free from putrescible matter. Enough fresh material was obtained from the post-mortem room to make examination of the soft tissues possible in addition.

GENERAL DESCRIPTION

The vertebral column shown in *Fig 597* presents features familiar to every museum student, and the features of the specimen shown in *Fig 598* are equally familiar to the radiologist or the pathologist. A knife thrust into an intervertebral interval of such specimens would meet bone. This event is to be expected from the findings by radiologists of fringes, of ledges, and of other bony projections from the upper and the lower edges of vertebral bodies which constitute 'lipping'.

The bony growths from macerated vertebræ have the following superficial characters —

1 Osteophytes commonly arise as spicules or as fringes of spicules from adjacent margins of vertebral bodies on their anterior or lateral aspects. I have never seen a vertebra bear osteophytes on the edge that is posterior and abuts on the spinal canal, and Beadle¹ makes the same observation.

2 Osteophytes usually bear longitudinal striations of ridges and grooves, best seen in osteophytes of moderate size.

3 Osteophytes are always broader at the base than at the point and spread over the anterior and the lateral surfaces, but not over the horizontal surfaces of the vertebral bodies.

4 Osteophytes may be larger, less diffuse, and nodular, and these may span an intervertebral interval. The outer surfaces of such nodules may be smooth or with striation retained at their bases only. Hemispherical, discoidal, or pyramidal forms may occur, and bizarre shapes also if adjacent nodules make contact.

5 Almost never are osteophytes to be found on a single unit of the vertebral column. Their incidence is spread over a group or over more than one group of adjacent vertebræ.

NOMENCLATURE

Chronic disease of the vertebral column has long received the name 'spondylitis deformans' when permanent changes in the bones and ligaments are associated with lasting alteration of posture—usually a kyphotic curve of the spine.

'Spondylitis', derived from *σπόνδυλος*, a vertebra, strictly refers to a unit of the vertebral column. The whole column, in its primitive form the ridged backbone of a quadruped, is *ράχτις*, whence is derived 'rachitis'. It would be misleading and pedantic to insist on the use of the term 'tuberculous spondylitis', though it would be more usually correct in both the pathological and the etymological sense. A disease spread widely over the vertebræ would be better rendered by 'rachitis' were not the word used as equivalent of 'rickets'. 'Senile rachitis' suggests a paradox, so perhaps 'polyspondylitis', indicating a plural incidence of disease

FIG. 597.—Photograph of a vertebral column affected with poly-spondylitis osteophytica. The presence of osteophytes on the upper and lower margins of the vertebræ is clearly shown. It is also to be noted that these osteophytes corresponds very nearly to that indicated in Fig. 601. Outcrops of osteophytes are to be seen in the cervical, the thoracic and the lumbar regions, but osteophytes are smaller or absent in the vicinity of the 2nd thoracic, the 12th thoracic, and the lower margin of the 5th lumbar vertebræ.

in the vertebræ, is to be preferred to the singular form 'spondylitis'.

Many authors use 'spondylitis deformans' to cover an imperfectly defined group of diseases which are all marked by new bone formation in the vertebral column. Usually 'spondylitis deformans' is described in the chapter or section devoted to 'osteo-arthritis'. Again, some authors use the term 'osteo-arthritis of the spine' to describe the disease which is the subject of the present note. The mutual growth of osteophytes from the bony elements of an intervertebral joint suggests a similarity to osteo-arthritis, which also is marked by bony growths from the margins of joints.

In common use 'osteo-arthritis' refers to a chronic disease of the so-called 'diarthrodial'



or 'synovial' joints, which are distinguished in every case by a cavity, by cartilage-covered contact surfaces, by a synovial membrane, and by a fibrous capsule. The joint between a pair of vertebral bodies has neither cavity nor synovial membrane. The flat ends of the vertebral bodies are partly covered by cartilage plates on which are built the intervertebral discs. These discs are composed of concentric plates of fibrous tissue which form a shell (annulus fibrosus) surrounding in the centre a mass of mucin-like material (nucleus pulposus) which is elastic, expansive, and retained under pressure. It is not to be expected that such a joint is subject to the same diseases as a synovial joint. Timbrell Fisher² has showed that true osteo-arthritis may be produced in the laboratory animal by methods which are quite inapplicable to a joint of any type but synovial.



FIG 598.—On the left is a drawing from a photograph of the middle part of the thoracic region of a diseased column. The specimen is viewed from the right anterior aspect, and the thickenings at the intervertebral intervals due to marginal osteophytes gives a moniliform appearance. There is less bulging in the region covered by the anterior longitudinal ligament, which is clearly indicated in the drawing. The bulging lies between the edge of that ligament and the back of the vertebral bodies in the region of the costo vertebral joints. In the middle of the specimen a bony nodule projects forwards and medially to overlap the front of the anterior longitudinal ligament. On the right is a reproduction of a radiogram of the same specimen taken in a lateral position. Lipping of the vertebral margins is clearly shown, especially at the prominent nodule in the middle of the specimen.

The osteophytes of osteo-arthritic joints, as seen in macerated bones, are the remains of cartilage-capped chondrosteophytes grown from the articular margins. In polyspondylitis marginalis osteophytica the osteophytes are osseous substitutions for ligament, and this probably accounts for the not uncommon occurrence of ankylosis, which is very rare in true osteo-arthritis.

Lawford Knaggs makes a welcome break with tradition when he gives a separate chapter to spondylitis deformans in his *Inflammatory and Toxic Diseases of Bone*. He distinguishes the disease whose features bear superficial resemblance to osteo-arthritis as 'spondylitis osteo-arthritis' from among other diseases peculiar to the vertebral column. This disease I confidently identify with polyspondylitis marginalis osteophytica, though Knaggs offers no exact description of its anatomy or distribution.

It is with much diffidence that one advocates the introduction of a fresh name into the vocabulary of pathology, especially when the name refers to a very common disease. 'Polyspondylitis' has been explained, and the justification of 'marginalis' will appear.

ANATOMY

1. Marginal osteophytes are not related to the attachments of muscles or of tendons. Though large nodules are found in the lumbar spine, and though fibres of the psoas muscle are attached alike to the ligaments which cover the nodules

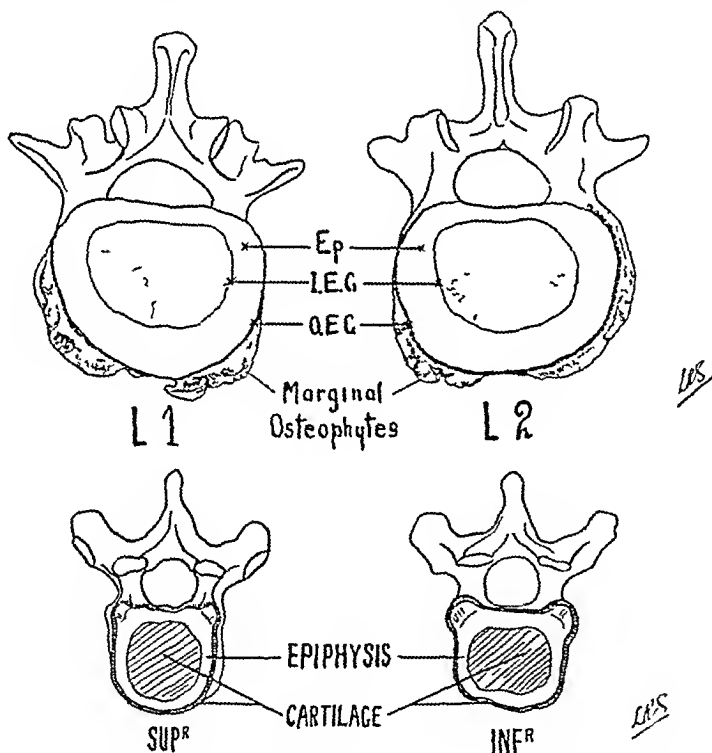


FIG 599—The two upper drawings show on the left the lower surface of the 1st and on the right the upper surface of the 2nd lumbar vertebra of the same column. Ep, Epiphysal ring, I.E.G., Inner epiphysal groove. The outer epiphysal groove (O.E.G.) becomes conspicuous in the presence of marginal osteophytes. The two lower drawings show the proportions of the epiphyses of the vertebral bodies in earlier life. The specimen, drawn both from above and below, is a thoracic vertebra from the skeleton of a male aged 16 years. The epiphyses, which bear the facets for the costal articulations, are embedded in cartilage. This cartilage appears between the edge of the epiphysis and that of the vertebral body, and also in the central part of the flat surface of the vertebral body.

and the normal parts of the lumbar spine, I have never observed an isolated tendon to be attached to a nodule. The same statement may be made of the muscle longus colli, though it must be admitted that nodules are rare in the cervical spine.

2 The osteophytes arise or 'break the surface' at a position on the vertebral body which is quite constant. If an affected vertebra is examined from the upper or the lower surface, it can be seen that the osteophytes are marked off from the body of the vertebra that bears them by a groove which marks their inner limits. It is by this arrangement that the word 'marginal' is justified. In Fig 599, which presents drawings of the flat surfaces of osteophyte-bearing vertebrae, these grooves are indicated, O E G representing 'outer epiphysial groove'.

The upper and the lower surfaces of the vertebral body from any part of the column bear a circumferential ring of slightly raised, flat, smooth bone. The inner edge of this ring is often irregular but contains an area which is porous in the macerated bone. In the fresh state this area is covered by a cartilage plate which closes the cancellous spaces of the vertebral body and abuts on the intervertebral disc. In early life this bony ring, which is the epiphysis of the vertebral body, is set in cartilage and falls short of the edge. At full growth the cartilage is ossified further and the ring reaches the edge. The inner edge of the ring is clearly defined

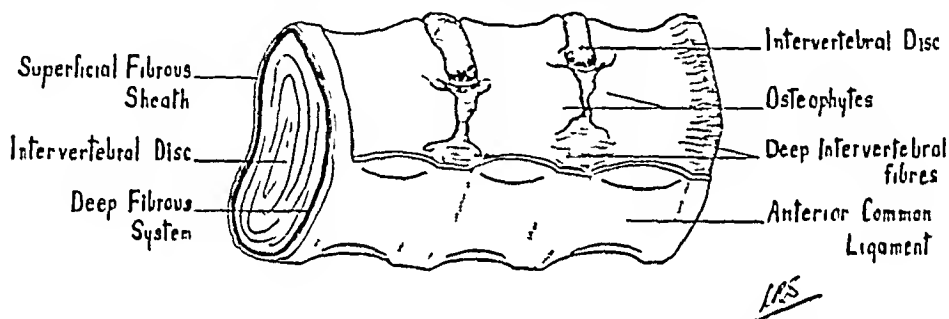


FIG 600—Shows in diagrammatic fashion the mode of formation of marginal osteophytes. A portion of a vertebral column is viewed from the lateral aspect. From the sides of the column the superficial lateral expansions of the anterior longitudinal ligament have been dissected away, but the front of the anterior longitudinal ligament is left intact. On the left a section has been made through the intervertebral interval and shows (a) the intervertebral disc with its sheath, (b) the deep intervertebral ligaments cut across and (c) the superficial fibrous system left as a cuff surrounding the column in the immediate vicinity of the section. After removal of the superficial fibrous sheath the deep intervertebral ligaments are displayed. On the right of the drawing these ligaments are shown in continuous series. In the other intervertebral intervals osteophytes are shown invading and replacing the deep intervertebral ligaments. Towards the top of the drawing are represented the sheaths of the intervertebral discs which come into view after removal of the deep fibres.

(NOTE—Anterior common ligament = anterior longitudinal ligament.)

and may be called the 'inner epiphysial groove'. Occasionally the outer edge of this ring may be traced in an adult bone as a very fine and narrow 'outer epiphysial groove'. It is when the osteophytes develop that the outer epiphysial groove becomes conspicuous, for a reason which will appear. The outer epiphysial groove receives the attachment of the sheath of the intervertebral disc.

3 *The Perivertebral and the Intervertebral Ligaments*—The whole vertebral column is enveloped in a continuous sheath of fibrous tissue. This conception, put forward by Macalister and confirmed by Poirier and Charpy, will be shown to be of some importance. The perivertebral sheath is weak at the sides, but medially, both in front and behind, specializes into anterior and posterior longitudinal ligaments. The fibres of these two ligaments are long, and pass over as many as three or four vertebrae. Deep fibres which span no more than one intervertebral interval lie between the intervertebral discs and the more superficial fibres.

These deep fibres are attached very firmly to the edges of the vertebral bodies. It is in these short deep intervertebral ligaments adjacent to the discs and outside

the outer epiphyseal groove that the osteophytes of polyspondylitis marginalis osteophytica arise, and it is by the spread of an ossile process into them that the outer epiphyseal groove is made conspicuous.

1. The superficial sheath of the vertebral column, of which the anterior common ligament is a component part, can be dissected away from the osteophytes quite readily, in spite of the partial blending of the fibres of the deep and the superficial systems. This point needs some emphasis because it has been claimed by Biddle that the longitudinal striations of the osteophytes "represent the fibres of the anterior common ligament, which over a long time has undergone a severe stretching force". In addition to the reason given above, it may be remarked that the osteophytes most commonly arise in a part of the circumference of the vertebral body which is lateral to the anterior common ligament; an example is shown in *Fig.* 598.

Fig. 600 shows the mode of formation of the osteophytes in schematic fashion. The drawing, based on the finding of a number of dissections, shows on the left a section made through an intervertebral interval. The ligaments, superficial and deep, are left as a cuff round the intervertebral disc. For the rest of the drawing, the superficial ligaments have been removed in a longitudinal strip and osteophytes are revealed lying in a deeper stratum, being embedded in the deep intervertebral fibres. In their turn these deep fibres have been removed in places to show the intervertebral discs and their sheaths.

DISTRIBUTION OF OSTEOPHYTES

The distribution of the osteophytes, determined from observations made on 106 vertebral columns, is shown in graphic form in *Fig.* 601. In collecting the data on which this graph is based the vertebrae were noted which bore characteristic osteophytes, and also the margins—upper, lower, or both—from which the osteophytes projected. The final count revealed inequalities in the total numbers of vertebrae, due to loss or damage for complete vertebral columns in a condition fit for examination are not too readily come by and therefore the results are presented in percentage terms. In the graph the osteophyte-bearing capacity of each vertebral margin is shown as if the total were 100.

The graph makes clear the following points in distribution:—

1. The liability of vertebrae to bear marginal osteophytes increases as the column is traced downwards.

2. There are three well-defined 'outcrops': (a) The *cervical outcrop* is marked off from the thoracic by a minimum zone at the junction of the 1st and 2nd thoracic units. (b) The *thoracic outcrop* is higher than the cervical, and is separated from the lumbar outcrop by a diminution zone at the 12th thoracic vertebra. (c) The *lumbar outcrop* is higher than the thoracic, but falls to a minimum at the lumbosacral junction. These three outcrops and minimum zones are shown in *Fig.* 597.

3. The osteophyte-bearing capacity of a pair of vertebral margins separated by the same intervertebral disc is not always the same. For example, the graph shows that more marginal osteophytes occur at the upper margin of the 2nd than at the lower margin of the 1st lumbar vertebra.

Commentary on Distribution—The zones of diminution show that weight in itself cannot explain the occurrence of marginal osteophytes. The weight of the trunk must increase from the 2nd to the 12th thoracic vertebræ and thence to the lumbo-sacral junction, but it is at these places that incidence is reduced.

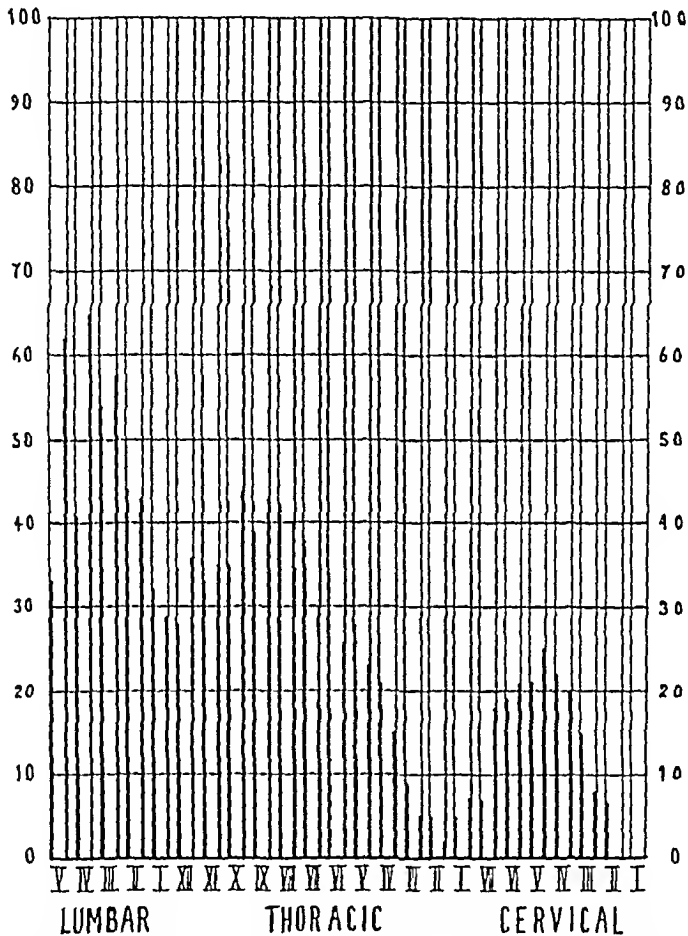


FIG. 601.—Graph showing the distribution of the osteophytes of polyspondylitis marginalis osteophytica in the human vertebral column. The abscissæ represent the several vertebræ set out in ascending order from left to right. Above the abscissæ are constructed upright rectangles whose right and left sides respectively represent the upper and the lower margins of the different vertebræ. Ordinates in heavy black on the sides of these rectangles indicate the numbers per centum of the upper or of the lower margins of the several vertebral bodies which have been found to bear osteophytes. The three waved form of the graph is to be especially noted, and the positions of the minimum points—at the 2nd and 12th thoracic vertebræ and at the lower edge of the 5th lumbar vertebræ. It will be observed that each wave increases in size as the column is traced downwards.

No author has described the curves of the vertebral column more carefully than has Humphry in *The Human Skeleton*. I have had the opportunity of examining Humphry's original preparations preserved at Cambridge and accept his conclusions *in toto*.

Humphry found that the mobile units of the vertebral column are arranged in three alternating antero-posterior curves, and that the junction points of these curves lie in the same vertical line with the line of gravity of the head and of the heads

of the femurs The reader may compare the points and regions of the vertebral column defined by Humphry with the significant points and regions of incidence of marginal osteophytes in the table set out below

ANATOMICAL OBSERVATION (<i>Humphry</i>)	SIGNIFICANCE	MARGINAL OSTEOPHYTES
Tip of odontoid process	= top of cervical curve	= zero
4th cervical vertebra	= zenith of cervical curve	= cervical maximum
2nd thoracic vertebra	= bottom of cervical and top of thoracic curve	= <i>diminution zone</i>
7th or 8th thoracic vertebra	= zenith of thoracic curve	= thoracic maximum, 7th to 10th vertebra
12th thoracic vertebra	= bottom of thoracic and top of lumbar curve	= <i>diminution zone</i>
	= ? zenith of lumbar curve	= lumbar maximum, 3rd to 5th vertebra
5th lumbar vertebra	= bottom of lumbar curve	= <i>diminution zone</i>

(The zenith of the lumbar curve is not stated by Humphry, who remarks that the curve is not the arc of a circle)

Evidently the explanation of the remarkable distribution of marginal osteophytes is to be found in the architecture of the vertebral column The diminution zones are at the balanced vertebræ through which the weight of the trunk falls vertically These vertebræ mark points of relative stasis in relation to the posture and the ordinary movements of erect-standing man For each curve, except the lumbar, the point of maximum incidence and the zenith have a near correspondence

CAUSATION

It has been shown that marginal osteophytes result from an ossific process in the deep intervertebral ligaments, and that it is the configuration of the vertebral column that determines the points of greatest and of least incidence

That pressure is a prime causative factor is shown by the changes that occur in the edges of a vertebra situated in the concavity of a scoliotic spine Vertebræ in such a situation become wedge-shaped, and on the side where compression is greatest develop flanges which have all the characters of marginal osteophytes In such vertebræ, however, the outer epiphysial grooves become obscured early Such a column as that shown in *Fig 604* results from some specialized attitude which has disturbed the whole balance of the column Apart from such an extreme, evidence of the predominant effect of mechanical influences is to be inferred from the distribution

Section of vertebral bodies with marginal osteophytes shows that the usual change is in the direction of increased density, and collapse of the bodies is rare It is to be deduced, therefore, that disease of the vertebral bodies is not the starting-point Evidence may, however, be adduced to suggest that the primary cause may be in the intervertebral discs The normal functions of an intervertebral disc include the maintenance of an expansive turgescient nucleus between each pair of vertebral bodies, to form a pivot for such rotation as vertebral bodies are capable of and to constitute about one-fourth of the height of the vertebral column

Independent research by Ross-Smith³ and by Beadle has proved that degenerative change in the nucleus of the intervertebral discs follows the fourth decade of life and is progressive thereafter An intervertebral disc without its turgescient nucleus, instead of keeping a pair of vertebral bodies in proper separation, is no

more than a mass of inelastic tissue to be pressed and moulded at the direction of the vertebræ between which it lies. It is possible that degenerated discs will spread under weight and exert pressure on circumferential ligaments by bulging them. Or vertebral bodies might slide or rotate on their neighbours and so throw strain on the intervertebral ligaments. That bulging of the intervertebral discs and of the overlying ligaments does occur is readily shown. *Fig 602* shows tracings of vertical sections through the same specimen as that illustrated in *Fig 598*. Further sections with similar findings are shown in *Fig 607*.

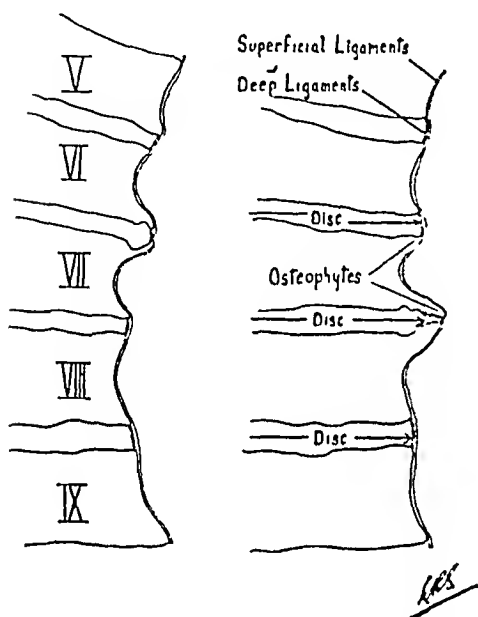


FIG 602—Tracings of sections made through the same specimen as is shown in *Fig 598*. In order to show the bulging of the intervertebral discs that underlie the osteophytes the specimen was cut with a fretsaw in two vertical planes. In each section the superficial ligaments that form the perivertebral fibrous investment, the deep intervertebral ligaments, and the outer limits of the intervertebral discs are shown in diagrammatic fashion. The tracing on the right traverses the conspicuous nodule shown in *Fig 598*. It will be seen that the intervertebral disc is bulged, the outer epiphyseal grooves are conspicuous, and the osteophytes are embedded in ligamentous tissue. A very small osteophyte is shown at the upper edge of the 7th vertebra, also accompanied by a slight bulging of the disc. The tracing on the left is in a plane slightly to the right of the other. Early osteophytes are shown growing from adjacent margins of the 6th and 7th vertebræ around the bulging intervertebral disc.

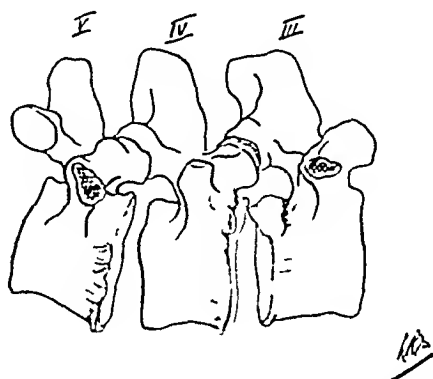


FIG 603—The three lumbar vertebræ in this drawing show an early stage in the growth of osteophytes from their margins. Distinct osteophytic flanges have begun to grow from the upper margins of the 4th and 5th lumbar vertebræ, while the lower margin of the 3rd and 4th are free. This mode of growth is usual in the lumbar column. The foramen between the 3rd and 4th vertebræ is partly obstructed by osteophytes. Growth of osteophytes in this position is uncommon, but might well be expected to cause a lesion of the 3rd lumbar nerve at its exit from the vertebral canal.

Sliding of vertebral bodies seems the most probable explanation of the phenomenon briefly referred to earlier, that osteophytic growth in the lumbar vertebræ is more extensive on the lower of an adjacent pair of margins than on the upper. The graph in *Fig 601* and the lumbar vertebræ shown in *Fig 603* illustrate this occurrence. This arrangement seems to be the outcome of the contours of the cervical and the lumbar

curves, in which the vertebral bodies lie in front of the line of gravity and weight bearing. In the thoracic curve, in which the vertebral bodies lie behind the 'plumb-line,' the same arrangement is found as high as the 9th vertebra or so, above that level the arrangement may be reversed.

In the lumbar curve, in consequence of the contour of the curve and the slope of the vertebral bodies, the weight of the column may thrust the disc forwards. This second factor, which is additional to the general bulging, affects only the

lower attachments of the ligaments and may account for the greater ossification in those positions

The longer anterior and posterior ligaments probably do no more than limit extreme dorsi- and ventri-flexion. Rotatory movements of vertebral body on vertebral body are limited by the deep intervertebral ligaments. Evidence that rotation does take place between vertebral bodies and is associated with the growth

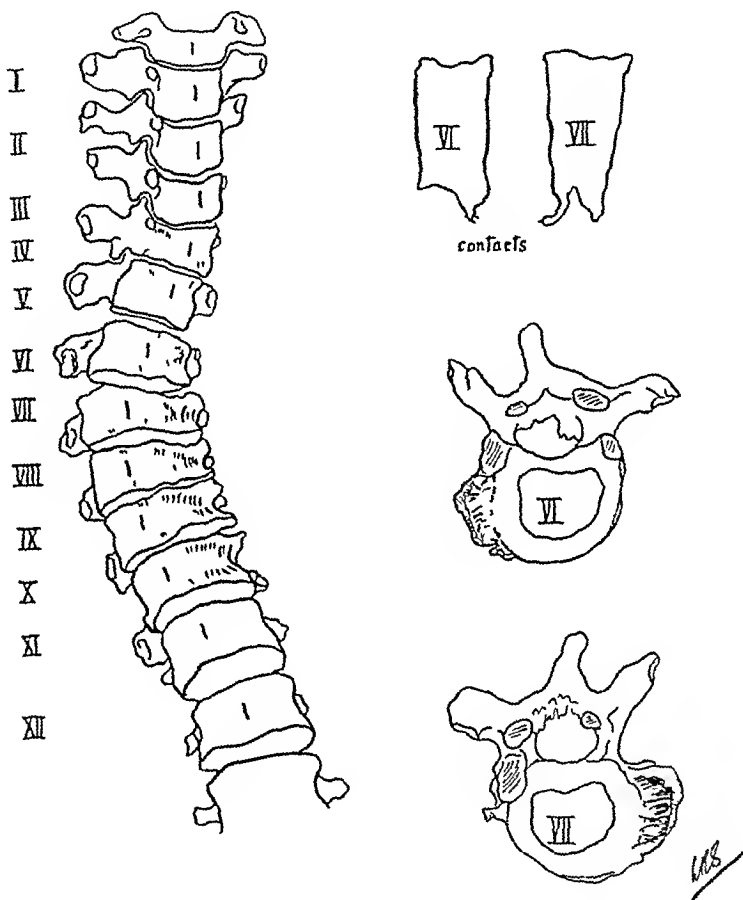


FIG 604—Drawings of certain specimens derived from a column which was the subject of scoliosis. The thoracic region, which was principally affected, is shown on the left in front view after reconstruction. The anterior mid-points of the vertebral bodies, determined by measurement, are shown by vertical lines. Evidently twisting has taken place between certain vertebrae, e.g., between the 3rd and 4th and between the 6th and 7th vertebrae. On the right above are shown the appearances of the 6th and 7th vertebrae in coronal section—note the wedge deformity of the 7th and the flangeing of both vertebrae. Below are drawings of the adjacent surfaces of the same two vertebrae. The osteophytic flanges arise outside the outer epiphyseal grooves in both specimens. Growth is evidently greater on the upper aspect of the 7th vertebra, in which the outer epiphyseal groove has become partly obliterated. The osteophytic growths from the 6th and 7th vertebrae made contact by areas which are distinguished by stippling in the two lower right-hand drawings.

of marginal osteophytes is not difficult to obtain. In Figs 604 and 605 the anterior mid-points of the vertebral bodies, determined by measurement, are marked by short vertical lines and make lack of alignment obvious. In the column shown in Fig 605 ankylosis has taken place between two vertebrae, and the rotation must have preceded ankylosis. Similar results are often obtained by measurement and reconstruction.

When the vertebral column begins to 'give' under weight, one curve may very probably rotate on the next and throw a strain on the deep intervertebral ligaments.

The 'anticlinal' vertebræ lie in the axis of rotation and suffer least from this effect. The further from the point of balance and the nearer the zenith of a curve, the greater is the excursion and the more intense is the strain in the ligaments.

A survey of the sheath of superficial ligaments that surrounds the deep ligaments and the intervertebral discs may explain the different degrees of osteophyte growth found in different parts of the circuit of the vertebral column. Osteophytes are uncommon on the front and unknown on the back of the vertebral margins, where the anterior and posterior longitudinal ligaments lie thick and strong. Laterally, the perivertebral fibrous sheath, of which the two long median ligaments are local thickenings, are thin and ineffective as supports to deeper structures. It is in the arc of the circuit of the vertebral margins between the two long ligaments that the majority of osteophytes arise. *Fig 598* shows that osteophytes may arise laterally to the anterior longitudinal ligament and grow to overlap the front of the ligament.

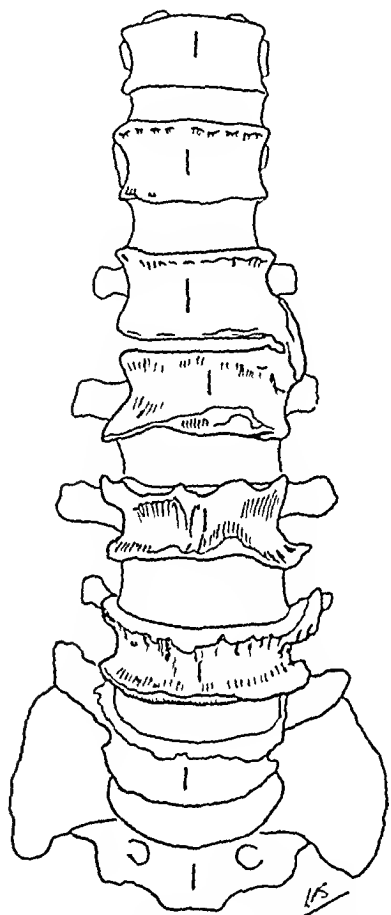


FIG 605—Anterior tracing of part of a reconstructed vertebral column showing the rotation that has taken place between vertebra and vertebra. The anterior middle points of the vertebræ, determined by measurement, are indicated by black vertical lines. Ankylosis has taken place between the 1st and 2nd lumbar vertebræ on the left side; lack of alignment of the middle points shows that twisting must have preceded ankylosis.

ANKYLOSIS

Many authors comment on the proneness to ankylosis in 'osteo-arthritis of the spine', and the rarity of the event in osteo-arthritis of ordinary synovial joints.

Fig 606 is a drawing of part of a vertebral column, and shows how osteophytes grow to meet each other from vertebra to vertebra in a strangely purposive manner. This need surprise us the less when we realize that ossific

substitution for ligament is taking place from each of its opposite attachments. Osteophytes, either by interlocking or by bony ankylosis, may form a splint and may set a limit to disease which is otherwise progressive, and may bring about spontaneous cure in which rigidity is effected at the cost of mobility.

This tendency to ankylosis has been exaggerated. Undoubtedly ankylosis takes place and commonly enough, but more often osteophytes with every superficial appearance of continuity make no more than close contact and interlock mechanically. A comparison of false and of true ankylosis is presented in two drawings in *Fig 607*.

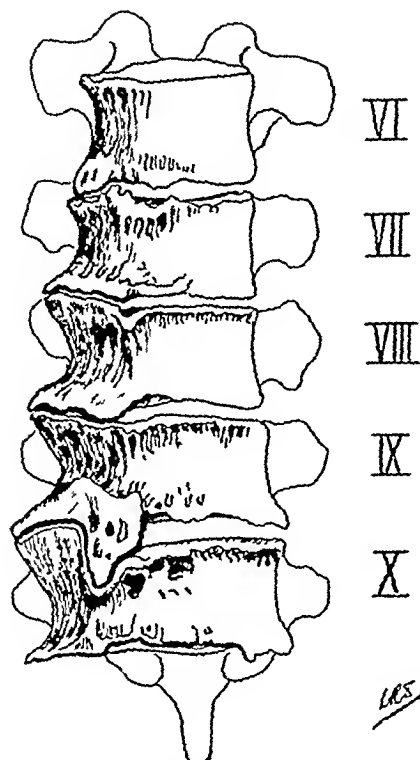


FIG. 606 —The vertebrae from the 6th to the 10th thoracic of the same column as is shown in Fig 597. The drawing is intended to show the 'purposive' fashion in which osteophytes grow towards each other from adjacent vertebral margins. Between the 9th and 10th vertebrae the osteophytes have developed an interlocking mechanism.

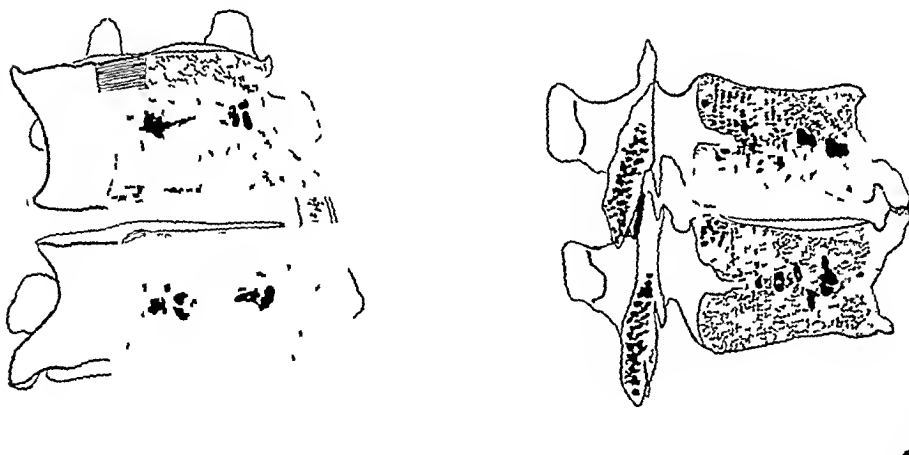


FIG. 607 —Drawings to contrast true and false ankylosis on the basis of internal structure. On the left is shown a section, drawn from a photograph, which passes through the 1st and 2nd lumbar vertebrae of the same column as is shown in Fig 605, after a wedge-shaped piece has been removed. It will be noted that the shell of compact bone is thicker at the pedestals of the bridge and the bridge itself than in the vertebral body. Strands of compact bone traverse the marrow cavity and disorganize the normal structure of the vertebra, but the marrow containing cavities are continuous from bone to bone. On the right is a section, also drawn from a photograph, which passes through 10th and 11th thoracic vertebrae whose ankylosis is false. The osteophytes from each of the vertebrae have made contact but not effected bony ankylosis. The osteophytes are composed of compact bone, are based on compact bone which traverses the two vertebrae that bear them in strands, but they do not yet contain marrow cavities. In both these sections note the bulging of the intervertebral space under the osteophytes, and the conspicuous outer epiphyseal grooves.

DEGENERATION OF THE INTERVERTEBRAL DISCS

The speculation has been made that degeneration of the nuclei of the intervertebral discs initiates the changes that end in *polyspondylitis marginalis osteophytica*. Strain in the deep intervertebral ligaments and osteophyte formation in them occur as the discs bulge and the vertebral bodies slide or rotate on each other.

The causes of degeneration of the intervertebral discs demand inquiry.

Arbuthnot Lane,¹ in his essays on labour changes, stated that as a consequence of carrying heavy loads, the vertebrae acquire density and the intervertebral discs are compressed and narrowed. Kaufman² has made the same observation more recently. On the evidence of radiograms Ross-Smith states that thinning of the intervertebral discs precedes the appearance at the vertebral margins of 'lipping', which may be identified with *polyspondylitis marginalis osteophytica*. It seems, then, that one cause of degeneration of the intervertebral discs may be traumatic, i.e., overweighting.

Ross-Smith also confirmed Beadle's observation that degeneration starts 'naturally' after the fourth decade of life and in its proportional incidence follows that of arteriosclerosis. A second cause of degeneration of the intervertebral discs, then, may be called 'senile'.

I surmise that a third cause may exist—namely a toxic one. There is need for research in making clear the physical path by which nutritive fluids reach the intervertebral discs. The cancellous marrow spaces of the vertebral bodies lack bony 'end-plates' and are closed by the cartilage plates with which the intervertebral discs are in contact. It is supposed that through these cartilage plates lymph-like fluid perfuses and bears the means of nutrition to the discs, which have no known blood-supply. If this be so, microbial or toxic invasion of a disc implies previous invasion of the vertebral body by microbes or toxins. It is further implied that the toxin invading the vertebral body is less virulent than that which causes osteoporosis and collapse, or else is selective of disc nucleus tissue. The resistance of the bone is likely to be better than that of the disc because of its blood-supply. The bone has to supply the osteoblastic cells which initiate osteophyte growth in ligament.

I can briefly outline the course of a case of severe 'lumbago'. X-rays showed narrowing of certain intervertebral discs and early growth of osteophytes from vertebral margins. A febrile illness and the known association of this change in the discs with typhoid infection led to a search for that organism. *B. typhosus* was not found, but a hæmolytic *B. coli* was isolated and a vaccine was prepared. The result of administration of the vaccine was striking, the patient's condition improved remarkably and a cure was effected. Subsequent X-ray examination showed the intervertebral discs to have recovered their proportions and the osteophytes to have become static. Such a case suggests that a toxic or a bacterial infection may cause *spondylitis marginalis osteophytica* secondary to disease of the intervertebral disc. It must be left for those with greater opportunities for clinical and pathological research to confirm or to reject the foregoing speculations.

SUMMARY AND CONCLUSIONS

Polyspondylitis marginalis osteophytica, or, more shortly in English, marginal spondylitis, is the familiar condition in which the vertebral bodies bear osteophytes at their margins or show 'lipping' in X-ray films.

The osteophytes are shown to arise at a constant position on the vertebral bodies, being separated from the flat surfaces of the vertebral bodies by grooves which mark the outer edges of the epiphyses. These grooves receive the sheaths of the intervertebral discs. The osteophytes themselves arise in the short deep ligaments that connect vertebral body to vertebral body. In consequence the osteophytes lie between the intervertebral discs and the superficial fibrous system which envelopes the whole series of vertebral bodies and includes the anterior and posterior common ligaments.

The distribution in the vertebral column of osteophyte-bearing vertebræ, when set out in graphic fashion, makes a three-waved curve with three districts of maximum incidence and three minimum points. The minimum points are at the 'anticlinal' vertebræ through which a plumb-line would fall in the erect attitude of the body. These vertebræ are supposed to be balanced and with a minimum tendency to slide or to rotate.

It is suggested that ossification is the result of strain put upon the short deep intervertebral ligaments when the nuclei pulposi of the intervertebral discs lose their normal turgid elasticity. Loss of turgescence permits intervertebral discs to bulge and vertebræ to slip or rotate upon their neighbours. Any of these events throw strain upon the deep intervertebral ligaments, and evidence of their occurrence is brought forward. The chain of circumstances that ends in the production of osteophytes is thought to commence in degeneration of the nuclei of the intervertebral discs. This degeneration may be brought about by trauma, i.e., by overweighing, by 'natural' senile change, or, perhaps, by the invasion of toxins.

The skeletons stored in the Anatomy Museum of the University of the Witwatersrand were those on which the first observations were made. I owe grateful thanks to Professor R. A. Dart and to the University of the Witwatersrand for the facilities afforded me. The work was continued on a large collection of vertebral columns in the Anatomy Museum at Cambridge. For opportunities of extended research my thanks are due to Professor J. T. Wilson and to Dr W. L. H. Duckworth. Dr A. Sutherland Strachan, Pathologist to the Johannesburg General Hospital, very kindly supplied the material from which I have studied the anatomy of the soft tissues of the vertebral column. Sir Walter Langdon-Brown, Regius Professor of Physic in the University of Cambridge, and the M.D. Committee permit me to publish these observations, which formed part of a thesis submitted for the degree of M.D. during 1933.

REFERENCES

- ¹ BEADLE, ORMOND A. *The Intervertebral Discs*, Medical Research Council Special Report Series No. 172, 1931.
- ² FISHER, TIMBRELL, *Chronic Non-tuberculous Arthritis*, 1929.
- ³ ROSS-SMITH, "The Intervertebral Discs", *Brit Jour Surg*, 1931, xviii, 358.
- ⁴ LANF, ARBUTHNOT, "Some Points in the Physiology and Pathology of the Changes produced by Pressure in the Bony Skeleton of the Trunk and Shoulder Girdle", *Guy's Hosp Rep*, 1886, xlii.
- ⁵ KAUFMAN, *Pathology*, 1929, 11.

EXPERIMENTAL SURGERY

SYMPATHECTOMY IN THE TREATMENT OF ACHALASIA OF THE CARDIA

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THE choice of a title for this paper has been somewhat difficult, as it appears that included amongst most series of cases of achalasia of the cardia or cardiospasm are three separate entities having a different pathological basis for their causation (1) Achalasia of the cardia, (2) Cardiospasm, (3) Hypertrophic stenosis of the cardia. The term 'achalasia' or absence of relaxation is therefore used in its widest sense to cover the common factor in all these three conditions—absence of relaxation of the cardia—and not to denote a definite pathological entity.

The clinical picture of achalasia of the cardia or cardiospasm is sufficiently well-known not to merit any further discussion in this paper. Reference must, however, be made to the nature of the obstruction met with. It is generally accepted that the most common site of the obstruction is at the level of the diaphragm. In a few cases, however, the dilatation may be seen to extend as far as the cardiac orifice of the stomach, as in three of Moore's¹ fourteen cases, and more rarely the obstruction may be well above the diaphragm (*see Fig. 612*). If the obstruction is due to the presence of an intrinsic sphincter, the whole lower inch or two of the œsophagus must partake of this sphincteric action, in order to account for the variable site of the obstruction. The presence of an anatomical sphincter is extremely difficult to demonstrate and has frequently been denied. However, Shattock² has shown two preparations which exhibit a widespread thickening of the circular muscle extending on to both œsophagus and stomach and situated entirely below the level of the diaphragm. Poulton and Payne³ describe a similar thickening in the lower portion of the œsophagus, and remark that it is noteworthy that that part of the œsophagus which partakes in the sphincteric action extends for some little distance above the diaphragm. It is of interest that in these observations the apparent sphincteric area surrounds a length of the lower portion of the œsophagus and is not sharply localized. In post-mortem examinations on typical cases of achalasia of the cardia or cardiospasm it is never possible to demonstrate any muscular hypertrophy in this sphincteric region. Despite the severity of the obstruction met with during life, the condition has been classically defined by Walton⁴ as one of "dilatation and hypertrophy of the œsophagus in which at post-mortem examination no obstruction can be found distal to the dilatation." Whatever it is that causes obstruction during life therefore ceases to act after death.

In contrast to this is a type of case described by Moore and Brown and Kellev⁵ which simulates achalasia of the cardia in its symptoms and X-ray appearances

(see Fig 616), but differs from this condition in that there is a muscular hypertrophy of the cardiac sphincter similar to that seen in congenital hypertrophic pyloric stenosis. In cases of this type, which are little recognized and are extremely rare, the obstruction does not disappear after death, but persists, so that it is possible to distend the œsophagus with water. The cardiac sphincter in these cases surrounds a narrow canal over an inch in length, the walls of which are greatly thickened. Such a condition can obviously not be due to any inco-ordination of the nervous control of the cardia, and stands in the same relationship to achalasia and cardiospasm as does congenital hypertrophic pyloric stenosis to pylorospasm, which latter conditions have been shown to be entirely separate entities.

If we leave aside this separate group of hypertrophic stenosis of the cardia and confine our attention to the true type as defined by Walton, we are immediately confronted with a variety of theories to account for the obstruction. These may be conveniently considered according to whether they postulate changes in the œsophagus itself or in adjacent structures. In the latter group Mosher⁶ believes that deformity of the œsophageal groove on the posterior surface of the liver alone or together with kinking of the œsophagus is responsible for the obstruction. Hill⁷ and Jackson⁸ regard an inco-ordination of the normal movement of the diaphragm during deglutition as the causative factor, Jackson postulating an actual phrenospasm, but the obstruction is not always at the level of the diaphragm, it may be above it or below. The recent work of Fulde⁹ is of interest in relation to this theory. This investigator describes an intrinsic sphincter mechanism in the lower portion of the œsophagus the action of which is normally modified by the phrenico-œsophageal membrane. This structure in virtue of its high attachment to the œsophagus closes the cardia by compression during descent of the diaphragm. To cause an obstruction of the œsophagus the diaphragm would therefore have to be fixed in the inspiratory position. Theories based upon changes occurring primarily in the œsophagus include the view expressed by Shaw and Woo,¹⁰ that kinking of the œsophagus produces the obstruction. This kinking is seen only in the more advanced cases where a combination of dilatation and elongation has deformed the œsophagus, and as this elongation results from the obstruction kinking cannot be the causative factor. As Walton has stated, the early cases are always of the fusiform type in which the cardia is in the most dependent position.

Finally, we come to those who believe that the obstruction is due to inco-ordination of the nervous mechanism of the cardia. Hurst¹¹ affirms that there is a paralysis of the vagal opening mechanism. Walton states that there is no paralysis but rather a spasm of the part (presumably he considered it as mediated through the sympathetic fibres), for which he suggests a congenital origin. The difference in their views is well illustrated in relation to the passage of bougies, for whereas Hurst maintains that a bougie will always pass the sphincter readily without resistance, Walton states that it is firmly gripped in its passage. For either of these views to be effective there must exist at the cardia a true intrinsic sphincter the tonus of which may be modified by the action of extrinsic nerves. A brief survey of the literature reveals a considerable discrepancy in the results obtained by experimental investigation of both these points. In a recent paper¹² the previous experimental evidence was summarized and the results of an investigation of the innervation of the œsophagus were published in full. The experiments were conducted on cats whose œsophagus as shown by Arey and Tremaine¹³ corresponds most closely in structure to the human

By stimulation experiments it was shown that the interdiaphragmatic and intra-abdominal portions of the œsophagus function as a true intrinsic sphincter which is relaxed by the vagus and contracts on sympathetic stimulation. The sphincter is capable of functioning independently of surrounding structures and receives its sympathetic supply from the cœliac plexus in fibres which follow the course of the left gastric artery and its œsophageal branch to the lower end of the œsophagus.

Excision of the extrinsic nerves supplying the œsophagus, as studied by X-rays taken of bismuth meals, showed that vagal excision reproduced the X-ray pathological and clinical picture of achalasia of the cardia. The sphincter failed to relax despite recovery of some peristalsis. These results were permanent, the changes produced being proportional to the extent of the denervation performed and the length of time elapsing after operation.

If, however, both thoracic sympathetic chains were excised at the same time as the vagi were divided, no obstruction resulted at the cardia, the sphincter showing



FIG. 608—X-ray showing the result of double vagotomy, seven months after operation. There is marked obstruction at the sphincter.



FIG. 609—X-ray showing relief of the obstruction at the sphincter following celiac sympathectomy. (Cf. Fig. 608.)

at first diminished tonus which later recovered somewhat but always relaxed to allow the meal to enter the stomach. Denervation of the sympathetic fibres which supply the cardiac sphincter by the operation of cœliac sympathectomy resulted in a complete loss of sphincter tonus, so that the meal could be caused to regurgitate from stomach to œsophagus. Later the tonus recovered to some degree but there was abnormally rapid passage of the meal through from œsophagus to stomach.

In animals in which achalasia of the cardia had been produced by a vagal section, subsequent cœliac sympathectomy resulted in a complete relief of symptoms, and X-rays now showed the sphincter relaxing and the meal entering the stomach without obstruction (*Figs. 608, 609*).

Animals treated by these means have now been followed for one year after sympathetic excision. There has been no onset of obstruction in animals with combined vagal division and thoracic sympathetic excision. In animals in which sympathectomy was performed after the development of achalasia there has been no recurrence of symptoms and a progressive gain in weight. X-rays still show the meal passing readily through the sphincter, with rather marked increase in the

peristalsis of the lower third of the œsophagus. The success of sympathectomy is to be judged on this relaxation of the sphincter rather than on regurgitation from the stomach to the œsophagus, which disappears as the muscle recovers tonus.

The bearing of these results upon the production of achalasia or cardiospasm is interesting. Bilateral vagal section if complete produces the symptoms and X-ray and pathological appearances of achalasia of the cardia. But if, however, the sympathetic fibres passing to the œsophagus are removed at the same time as the vagal section is performed, no obstruction results, and following the production of the obstruction it may be relieved by sympathectomy.

It follows, therefore, that whether we subscribe to the views of Hurst, that the obstruction is due to vagus failure, or those of Walton, that it is due to a spasm, in both cases the integrity of the sympathetic supply is necessary for the obstruction to occur, and therefore it should be relieved by sympathectomy.

CLINICAL PATHOLOGY

The study of 66 cases in various hospitals has shown that amongst them are some in which a bougie passes the sphincter readily despite severe clinical and X-ray evidences of obstruction, presumably an achalasia or vagus failure. In others of the spasmodic type the bougie is firmly gripped in passing, and in these the symptoms sometimes show a characteristic intermission. It is therefore felt



FIG. 610.—Section of lower end of œsophagus from a case of achalasia or cardiospasm. There is superficial œsophagitis. The region of Auerbach's plexus is free from inflammatory changes.

that all cases of achalasia have not the same pathogenic factor and that it is desirable to separate them into three groups: (1) Cases of vagus failure—achalasia, (2) Cases of spasmodic contraction (cardiospasm), (3) Hypertrophic stenosis of cardia. The latter is not truly an example of achalasia for the reasons already stated, but, being extremely liable to be mistaken for it, it is included here for the sake of further emphasis.

Under the first group, lesions of the vagus trunk are rare. Kraus¹¹ has described a case in which the vagus nerve was degenerated. In another Politzer¹⁵ described involvement of the vagus by a mass of glands. Although isolated cases of trunk lesions are described, the main site of vagus involvement is at the ganglia of Auerbach's plexus, in which Rake¹⁶ has demonstrated chronic inflammatory changes. These show a progression from round-celled infiltration to degeneration of ganglion cells and ultimately to complete fibrosis. The importance of these changes is, that these ganglion cells constitute vagal relays, a view held by such authorities as Catherine Hill,¹⁷ Kuntz,¹⁸ and Abel.¹⁹ Dr Rake has allowed me the privilege of examining his preparations, and the changes shown in these are most striking.

Cases, however, occur which do not show degenerative changes. An example of this type is shown in *Fig 610*, obtained from a woman who died during a thyroid crisis but had incidentally achalasia. In this case there is a superficial œsophagitis, but the region of Auerbach's plexus is not affected. This merely shows that all cases have not a common cause, without in any way disproving a very definite factor in the production of the disease. The changes in Auerbach's plexus, as pointed out by Gask and Ross,²⁰ may have a definite bearing upon the treatment of this condition by sympathectomy. "Should it be established, as is commonly supposed, that an inflammatory or degenerative lesion of Auerbach's plexus is invariably present, division of the sympathetic supply proximal to Auerbach's plexus would not be expected to be beneficial." Alternatively, if the normal function of Auerbach's plexus is interfered with, sympathectomy, while producing diminished sphincter tonus, might still leave the tube dilated.

In considering cases of spasmodic type Walton, in his Jacksonian Essay on cardiospasm, discards cases of hysterical spasm in young women, as these fail to show the dilatation so characteristic of the disease. Similarly cases of local reflex spasm from such causes as œsophagitis, peptic ulcer, and following upon such operations as gastro-enterostomy, although of interest, are usually transient or disappear on the cure of the causative lesion.

True examples do, however, recur. One case to our knowledge succeeded upon a perineal excision of the rectum and presented marked symptoms. In another (*see Fig 612*) there was a recurring and extremely severe spasm attended by dilatation of the œsophagus in a young man following upon a perforated duodenal ulcer, and in this patient the bougie could be felt to be firmly gripped in its passage. Walton goes further and considers that the majority of cases are due to spasm.

TREATMENT

The various forms of treatment at present used are summarized here, and are sufficiently familiar to need but little amplification.

- | | |
|---------------------------------------|--|
| 1 Dilatation of the cardia | { Hurst's mercury bougie
œsophagoscopy and bougie
Hydrostatic dilators
Digital dilatation |
| 2 Plastic operations on the œsophagus | { Reisinger
Freeman |
| 3 Plastic operations on the cardia | |
| 4 Short-circuit operations | |

For dilatation of the cardia Hurst advocates the passage of a rubber bougie of large diameter weighted with mercury which he says in all cases passes the sphincter readily, and at one time stated that the condition could always be relieved without operation. Alternatively a solid bougie may be passed from the mouth under direct vision by œsophagoscopy. The passage of such an instrument blindly cannot be too strongly condemned. Various forms of hydrostatic dilators, such as those of Gottstein, Plummer, and Mosher, have been devised. These consist essentially of a hollow tube with an inflatable bag surrounding its terminal portion. This may be introduced into the cardia and distended. The degree of stretching is controlled by the incorporation of a manometer in the system if water is used. Alternatively the system may be air-containing, and the sense of resistance used as an indication of pressure exerted. A more extensive dilatation is secured by the operation of gastrostomy and digital dilatation in which the cardia may be sufficiently stretched to accommodate three fingers. Plastic operations on the œsophagus are of historic interest. They have been designed to correct either the dilatation (Reisinger) or the lengthening of the œsophagus (Freeman). Plastic operations on the cardia are of two types. In the first, which is similar to the Heineke-Mikulicz pyloroplasty, a longitudinal incision is made and closed transversely. Heller's incision is similar to Rammstedt's operation for congenital hypertrophic pyloric stenosis. But since in typical cases there is no muscular hypertrophy of the cardia this procedure appears to be ill-founded. Short-circuit operations consist essentially in the formation of an œsophago-gastrostomy either above or below the diaphragm.

In order to arrive at an assessment of the value of these methods in general hospital practice an inquiry was undertaken. Sixty-six cases were traced and their present condition ascertained. As some patients had more than one form of treatment 85 results are available for comparison. It is not suggested that these are at all typical of the results to be obtained by dilatation in cases of a mild degree. All the records were obtained from surgical wards, and therefore presumably include the more severe variety.

TYPE	NO	CURE	IMPROVEMENT	MORTALITY
Mercury bougie	28	2	8 cases	—
Œsophagoscopy and bougie	16	—	Up to 1 year	3 (20 per cent)
Dilators	5	—	7 months to 1 year	—
Digital dilatation	26	8	2 months to 3 years	3 (11 per cent)
Heller's incision	2	—	1 month to 3 months	—
Cardioplasty	1	—	1 year	—
Gastrostomy	5	—	—	—
Œsophago-gastrostomy	3	3	—	—

Treatment by Hurst's bougie gave a complete cure in 2 cases, 8 were much improved, with gain in weight. In the remainder the relief was incomplete, the symptoms recurring if the bougie were discarded, the tube being used as often as two to three times a day. Those patients who did not find passing the tube impossible complained of the inconvenience or depression caused by its repeated passage, and in some cases of the attacks of œsophagitis which this produced.

Dilatation by bougies resulted in three deaths, two from empyema and one from perforation of the stomach. As this occurred even under direct vision it is a strong point against the blind passage of an œsophageal bougie. Gastrostomy and digital dilatation was completely successful in eight cases up to the time of inquiry (3 to 7 years). The remainder showed recurrence of symptoms in periods varying from two months to three years after operation. Although the recurrent symptoms were less severe in some cases, they nevertheless required further treatment by a bougie. The fact that gastrostomy alone was performed in 5 cases shows that contrary to Hurst's statement it is definitely impossible to pass the sphincter in certain cases. Three cases of œsophago-gastrostomy all showed complete relief on a slightly restricted diet after three years. It appears from these figures that the various forms of treatment by dilatation are not completely successful. The period of relief appears to be proportional to the initial degree of stretching, being longest in the case of digital dilatation. Plastic operations have little to recommend them, the most uniformly successful operation is œsophago-gastrostomy, which is a procedure of some magnitude.

In the treatment of this condition by sympathectomy, Recalde²¹ described an operation designed to remove Auerbach's plexus, which he regards as responsible for maintaining the obstruction. To effect this, the external muscular coat was stripped from the sphincteric area and the underlying plexus destroyed. Such a procedure must also interrupt the sympathetic supply to the circular muscle of the cardia. Of the 4 cases in his series, 1 died from perforation, but 3 remained completely relieved of their obstruction. Although these results encourage the possibility of sympathectomy, the method has none of the advantages of a remote neurectomy. From the experimental findings, it has been shown that the sympathetic supply to the cardia may be approached either by excision of the lateral thoracic sympathetic chains or by interruption of the fibres along the course of the left gastric artery. Kuntz²² states that in man the œsophagus is innervated chiefly from the inferior cervical ganglia, further branches also passing to the œsophagus from the thoracic sympathetic ganglia. Hovelacque²³ considers that these thoracic branches are few in number and not to be found in all subjects. Where they occur they are more numerous above the origin of the great splanchnics. It is probable that certain of these fibres may in part contribute to the sphincteric supply through the pericœsophageal plexus. Their denervation could be effected by a bilateral cervico-thoracic ganglionectomy designed to include the upper thoracic ganglia. Since such a procedure interrupts the sympathetic supply to the head, upper limbs, and heart, with the production of a bilateral Horner's syndrome, it appears to be a mutilating procedure. The report of one case treated by this means has been made recently from the Mayo Clinic²⁴. The patient obtained symptomatic relief, but a bilateral Horner's syndrome was produced. An X-ray of the condition of the œsophagus following operation is not published. Denervation of the left gastric artery appears to be preferable provided that a sufficient denervation is obtained. Evidence that this operation affects the human cardia as in cats was obtained from a case of celiac sympathectomy, the first on record, performed by Mr J B Hume,²⁵ of St Bartholomew's Hospital, in the treatment of gastric ulcer. Two years after operation this patient was recalled and X-rayed. There was seen to be abnormally rapid passage of the meal through the cardia.

Dissection in the human shows that in order to interrupt the fibres passing

to the cardia, it is not necessary to strip the trunk of the cœliac axis artery. The cardiac sphincter supply is derived chiefly from the left side of the cœliac plexus. Fibres from the right side are distributed mainly along the hepatic branch, a few pass along the left gastric artery.

From the cœliac plexus fibres pass in a broad leash towards the left gastric artery, joining at various levels along its course and forming a network in the surrounding fat, the majority lying posterior and to the right of the vessel. From this network two main divisions form, which are distributed with the left gastric artery to the stomach and with the œsophageal branch to the cardia. This distribution is shown in *Fig 611*, a drawing made of the portion of tissue removed in the course of operation. To effect a denervation it is necessary to excise the left gastric artery with its surrounding fat and nervous tissue. Division of the artery alone

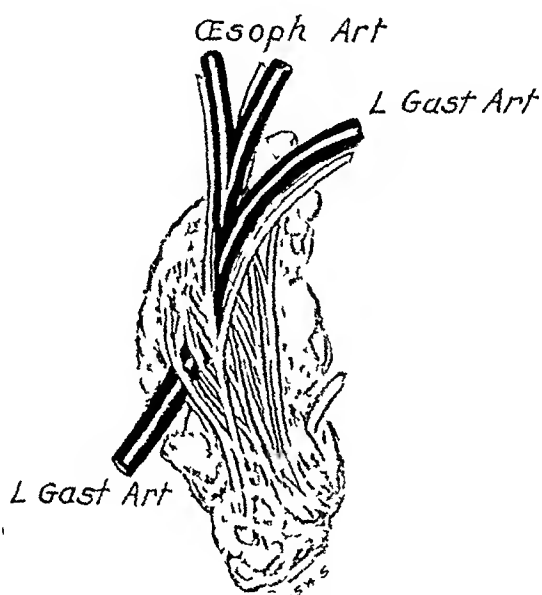


FIG 611.—Drawing of portion of tissue excised at operation. This shows the large number of fibres passing to join the left gastric artery, forming a plexus on its anterior and posterior surface. The majority of the fibres pass posteriorly.

is obviously inadequate. Division of the trunks alone fails to interrupt the periarterial network. Rarely a branch may be seen as described by Brandt,²⁶ which passes directly from the left cœliac ganglion to the cardia and not along the course of the vessels.

The approach to these structures is made through the lesser omentum, and in all cases it is necessary to excise the left gastric vein in order to obtain an exposure. The incision may be a left paramedian, or, as illustrated in *Fig 616*, a double subcostal incision may be employed. The latter incision gives an excellent exposure.

During August, 1934, a suitable case for operation was found in the wards of the Royal Infirmary, Edinburgh. Mr W A D Adamson²⁷ carried out the operation. Since that time he has operated on three cases. For his readiness to

apply this new operation and his generous collaboration in keeping me informed of his results, I am deeply indebted

It was not until November, 1934, that the first suitable case became available in the Surgical Unit at St Bartholomew's Hospital, London. Professor G. E. Gask undertook the operation, and has since operated on three additional cases. I am deeply grateful for his continued advice and suggestions during the course of this work.

The cases described are not intended to form a complete series. Three examples (one of each of the three types described on p. 867) are chosen to form a preliminary report.

CASE REPORTS

Case 1—Male, aged 27. In November, 1933, suffered from a perforation of a duodenal ulcer. Convalescence complicated by rupture of the abdominal wound. In May, 1934, he returned complaining of persistent vomiting. He was emaciated and had acetone breath. X-rays showed *spasmodic contraction of the lower œsophagus with obstruction* above the level of the diaphragm. He was treated on three occasions by œsophagoscopy and the passage of bougies, which were felt to be firmly gripped at the sphincter. The relief



FIG. 612—*Case 1* (Mr. Adamson's)

of the œsophagus with typical cone shaped termination at its lower end



FIG. 613—*Case 1* After operation (Dec. 12, 1934) eight seconds after swallowing. Shows the meal passing readily through the site of previous obstruction. The dilatation has subsided.

afforded by this means showed a progressive decrease on each occasion from three weeks to one week, and on the last occasion to four days. On August 4 operation was performed, his weight then being 9 st. 2 lb. Since operation he has remained perfectly well and can eat anything. His present weight is 10 st. 8 lb. *Fig. 612* shows the œsophagus before operation, twenty minutes after swallowing. *Fig. 613* shows the œsophagus five months after operation, eight seconds after swallowing. The meal can be seen passing readily through the previously obstructed segment.



FIG 614—Case 2 (Mr Adamson's) X-ray before operation (Nov 8, 1934), twenty minutes after swallowing. Shows obstruction at the level of the diaphragm, with dilatation of the œsophagus.



FIG 615—Case 2 After operation (Dec 20, 1934), eight seconds after swallowing. Shows diminished sphincter tonus with the meal passing into the stomach. There is still some dilatation.



FIG 616—Case 3 (Professor G E Gask's) X-ray before operation (Nov 14, 1934), ten minutes after swallowing after the previous removal of 8 oz of residual fluid. Shows dilatation of the œsophagus with obstruction below the level of the diaphragm.



FIG 617—Case 3 X-ray six weeks after operation, ten minutes after swallowing. The dilatation and residual fluid were diminished, but obstruction still persists at the cardia.

Case 2—Male, aged 28 April, 1934 Following upon pneumonia he developed symptoms of vomiting and dysphagia X-rays on Nov. 8 showed the œsophagus to be dilated, with obstruction at the level of the diaphragm As a diagnostic procedure bilateral injection

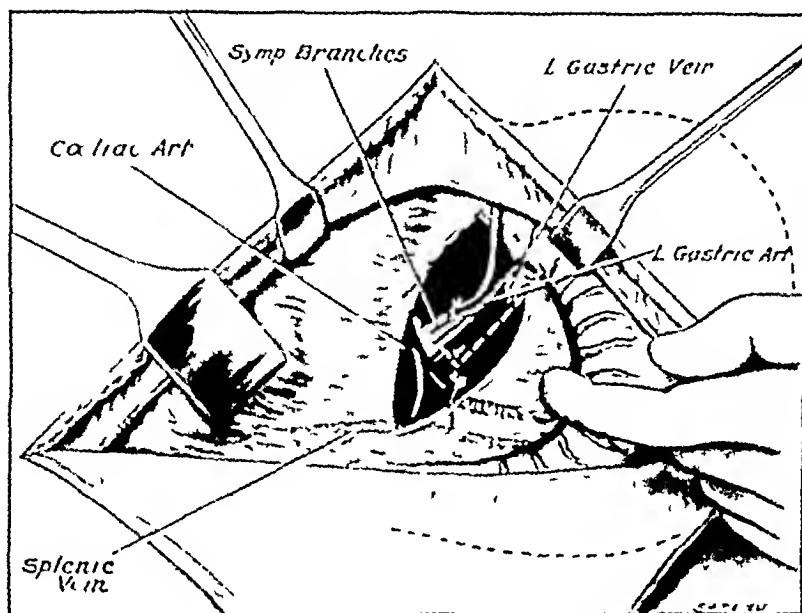


FIG 618—*Case 3* Operation double subcostal incision The left lobe of the liver has been mobilized and retracted to the right The lesser omentum has been incised and the left gastric vein divided The left gastric artery is divided at its origin The portion of tissue to be excised is included in the white frame

of the stellate ganglia was performed and was seen to have no effect upon the outline or peristalsis of the œsophagus Operation was performed on Nov. 12 Since operation swallowing has improved and he is gaining weight Fig. 614 shows the condition before operation, twenty minutes after swallowing Fig. 615 shows the condition after operation, eight seconds after swallowing There is diminished sphincter tonus and the meal is passing into the stomach

Case 3—Female, aged 47 Seven years' history of dysphagia and vomiting Treated for five and a half years by the regular passage of bougies without permanent relief Loss of weight from 16 stone to 11 st 10 lb X-ray (Fig. 616) after the removal of 8 oz of residual fluid from the œsophagus showed marked dilatation, with obstruction below the level of the diaphragm The meal was held for half an hour with the passage of only a minute quantity to the stomach Operation on Nov. 20, 1934 A double subcostal incision was employed (Fig. 618) and the left gastric vein divided The left gastric artery was excised from its origin to the termination of the œsophageal and gastric branches, together with surrounding fat and nervous tissue (Figs. 619, 620) Palpation of the cardiac region showed that the sphincter was enormously thickened,

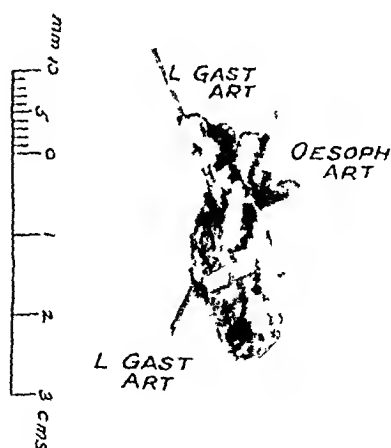


FIG 619—*Case 3* Photograph of a dissection of the tissue excised at operation posterior aspect There has been some shrinkage owing to fixation

approximately the size of a man's thumb, and over an inch in length. During this palpation the thickened area was felt to relax twice in the course of one minute at the close of the operation. After operation swallowing was improved, the patient taking a full diet regularly without regurgitation. There was, however, an occasional sense of obstruction, but this was less marked. X-rays six weeks after operation showed diminished dilatation of the œsophagus, but there is still obstruction at the cardia (*Fig 617*). The sense of obstruction is now increasing.



FIG. 620.—Case 3. Section of the tissue excised, showing a large number of non-myelinated trunks surrounding an artery. It will be noticed that there is a concentration of the nerve trunks in one area, actually posterior and to the right of the vessel.

The first case is of spasmodic type and shows complete relief after neurectomy. The second case appears to be a true achalasia, bilateral injection of the stellate

ganglia did not affect the peristalsis of the œsophagus. This case shows diminished sphincter tonus, but there is still some dilatation. The third case is an example of hypertrophic stenosis of the cardia. As the obstruction in this condition persists after death it could not be completely relieved by neurectomy.

SUMMARY

1 Included amongst most series of achalasia of the cardia or cardiospasm are three conditions, true achalasia, cardiospasm, and hypertrophic stenosis of the cardia.

2 The first two result from autonomic imbalance in the control of the cardia and are improved by sympathectomy. The third stands in the same relationship to achalasia or cardiospasm as does hypertrophic pyloric stenosis to pylorospasm. It is a separate entity and shows little improvement following sympathectomy.

3 The operation of left gastric sympathectomy affords relief in cases of the first two types.

4 Details of the operation are given, and the results of the operation in cases of each type are described.

I wish to express my thanks for permission to use cases from Professor Gask's and Mr Adamson's series.

REFERENCES

- ¹ MOORE, *Proc Roy Soc Med (Sect of Laryngol)*, 1918-19, vii, 68
- ² SHATTOCK, *Ibid*, 92
- ³ POULTON and PAYN, *Quart Jour Med*, 1923-4, vii, 60
- ⁴ WALTON, *Brit Jour Surg*, 1924-5, vii, 701
- ⁵ BROWN and KELLEY, *Proc Roy Soc Med (Sect of Laryngol)*, 1918-19, vii, 48
- ⁶ MOSHER, *Laryngoscope*, 1922, xxxiii, 348
- ⁷ HILL, *Proc Roy Soc Med (Sect of Laryngol)*, 1918-9, vii, 37
- ⁸ JACKSON, *Laryngoscope*, 1922, xxxiii, 139
- ⁹ GULDE, *Deut Zeits f Chir*, 1924, ccxlii, 580
- ¹⁰ SHAW and WOO, *Proc Roy Soc Med (Sect of Med)*, 1916, Dec, 1
- ¹¹ HURST, *Quart Jour Med*, 1930, xliii, 491
- ¹² KNIGHT, *Brit Jour Surg*, 1934, vii, 155
- ¹³ AREY and TREMAINE, *Anat Record*, 1933, lvi, 315
- ¹⁴ KRAUS, *Nothnagel's Spec Path u Therap*, 1902, 129
- ¹⁵ POLITZER, *Munch med Woch*, 1913, l, 108
- ¹⁶ RAKE, *City's Hosp Rep*, 1927, lxxvii, 141
- ¹⁷ HILL, C, *Trans Roy Soc (Sect B)*, 1927, ccvi, 355
- ¹⁸ KUNTZ, *Anat Record*, 1922, lvii, 193
- ¹⁹ ABEL, *Jour Anat and Physiol*, 1913, lvi, 35
- ²⁰ GASK and ROSS, *Surgery of the Sympathetic Nervous System*, 124
- ²¹ REGALDE, *Arch ital di Chir*, 1932, xxxv, 613
- ²² KUNTZ, *Autonomic Nervous System*, 1929, 1st ed, 188
- ²³ HOVELACQUE, *Anatomie des Nerfs*, 1927, 1st ed, 720
- ²⁴ CRAIG, MOERSCH, and VINSON, *Proc Mayo Clin*, 1934, Vol i, 749
- ²⁵ HUME, *St Bart's Hosp Rep*, 1933, lvi, 28
- ²⁶ BRANDT, *Zeits f angewandte Anatomie und Konstitutionslehre*, 1919-20, 302
- ²⁷ ADAMSON, *Proc Roy Soc Med (Sect of Surg)*, 1935, January

SHORT NOTES OF RARE OR OBSCURE CASES

BLACK HAIRY TONGUE OR HYPERKERATOSIS LINGUAE

BY SURGEON CAPTAIN H E R STEPHENS, OBE, RN

ON Sept 11, 1934, a Regulating Petty Officer belonging to H M S *Dolphin* was admitted to the Royal Naval Hospital, Haslar. A week previously he had noticed a tuft of black hair on the back of his tongue. This was discovered quite by chance and did not cause any inconvenience. On examination there appeared to



FIG 621—Showing condition as excised (2)

be a ridge of hair on the dorsum of the tongue commencing just in front of the foramen cæcum and circumvallate papillæ, this extended forwards for $1\frac{1}{2}$ in being about $\frac{3}{8}$ in wide at its broadest part. On Sept 14 the growth was excised by Surgeon Commander E Heffernan. The patient was discharged to duty on Oct 5, completely cured.

The accompanying microphotographs (Figs 621-623) were taken from the excised specimen by Surgeon Commander J A O'Flynn. These show the condition better than any written description. The pathology appears to be obscure, some authorities believe that an infective process is the causal factor, while others

consider it to be a simple hypertrophy with pigmentation of the lingual papillæ. The best account of this interesting disease is given by Andrews¹ and is quoted in full.

"The affection is rare, usually occurring on the dorsum anterior to the circumvallate papillæ, where there are formed black, bluish-black, or brown patches



FIG 622 —Section of excised growth (4)



FIG 623 —Section shown in circle in Fig 622 stained by magenta red and picro-indigo carmine (\ 30)

consisting of hairlike intertwining filaments from $\frac{1}{4}$ to $\frac{1}{2}$ in in length. Histologically, Heidingsfeld found elongated and stratified filaments originating from abnormal papillæ within the epithelial covering of the mucous membrane. The cells making up the hairlike processes resemble those on the stratum corneum of

the skin. The colour is due to changes in the horny cells analogous to those which are seen in certain types of hyperkeratosis of the cutaneous surface, or to pigment from substances taken into the mouth. The condition is not due to micro-organisms, but to some congenital abnormality which develops in later life.

"Pseudoblack tongue may originate from local irritants or mouth washes containing coloring matter. Oppenheim demonstrated that hypertrophy and hyperkeratosis of the filiform papillae in this region can be experimentally produced by daily applications of tinctures containing vegetable coloring matter. He compares the artificially produced condition of the tongue with hyperkeratosis of the skin from tar and aniline products. Prinz believes that the pigmentation of the elongated filiform papillae is always due to a deposit of pigment from external sources. He was able to demonstrate iron pigment on the filaments of black tongue by chemical tests, and believes the condition is due to a chemical reaction between the hæmoglobin in the blood and certain sulphur and ammonia compounds derived from the decomposition of protein debris or tobacco.

"Another form of pseudoblack tongue is caused by the filamentous mycelia of fungi which grow as molds on the dorsal surface. The diagnosis is confirmed by microscopical demonstration of the fungus and by the ease with which the filaments can be removed from the tongue. Thompson and Montgomery, in a study of two cases, isolated pure cultures of actinomycosis from each. Weidman recovered a similar organism (*Micosporon minutissimum*) in a case of black tongue occurring in a diabetic who also had erythrasma."

REFERENCE

¹ ANDREWS, G. C., *Diseases of the Skin*, 1930, 992. Philadelphia and London.

A RARE TYPE OF VALVULAR OBSTRUCTION OF THE NECK OF THE BLADDER RECORD OF TWO CASES

By J. R. LEARMONTH

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AND KENNETH H. WATKINS

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HONORARY ASSISTANT SURGEON, MANCHESTER NORTHERN HOSPITAL

THE presence of valvular folds at the neck of the bladder is an uncommon cause of urinary obstruction, although a number of cases are on record in which valves partially blocked the posterior urethra. In cases of this type a diagnosis is rarely made until prolonged back-pressure and infection have produced irreparable damage to renal function. Of the recorded cases in which the obstructing tissue was wholly intravesical, in that of Harris¹ the valve arose from the posterior margin of the vesical orifice, in that of Campbell² a large fold, termed by him the "trigonal curtain", arose midway between the vesical outlet and the inter-ureteric ridge, while in Wallace's³ case a fibrous partition or diaphragm arose from the

inter-ureteric ridge In the two cases we are to describe the obstructing fold was attached to the anterior margin of the vesical orifice, an arrangement which, so far as we are aware, has not hitherto been recorded

Case 1 (J R L)—A man aged 40 years was admitted to the Aberdeen Royal Infirmary on Sept 2, 1933 He stated that for fifteen years he had had difficulty in passing urine This was most marked at the beginning of the act, once started, the stream passed fairly easily On two occasions during this period he had been catheterized to relieve complete retention Occasionally there had been a scalding sensation during the act of micturition In other respects he had always considered himself to be healthy

ON EXAMINATION—The patient was a healthy and well-developed man The tongue was clean, the throat not inflamed There was no evidence of disease of the nervous system, of the lungs, or of the cardiovascular system The bladder was distended to an inch below the umbilicus, otherwise abdominal examination was negative On rectal examination the prostate was small and soft, and the tone of the anal sphincter good The urine was alkaline and contained albumin, there was a copious deposit of pus X-ray examination did not disclose any calculus or any osseous deformity of the spine

ON ADMISSION—The value for blood-urea was 265 mgrm per 100 c c The Wassermann reaction in the blood was negative An ordinary rubber catheter could be passed with ease, so that the 'admission' diagnosis of stricture of the urethra was obviously incorrect

For a week he was regularly catheterized three times a day, from 300 to 400 c c of urine being withdrawn at each session, in the intervals he passed urine in amounts of 120 to 240 c c, so that the total daily output was about 2000 c c Thereafter, an indwelling rubber catheter was employed, and infusions of 10 per cent glucose solution were given by the intravenous route Five days after the introduction of the catheter, on Sept 15, the patient complained of sore throat, and the temperature rose This condition appeared to be subsiding, and by Sept 20 the value for blood-urea was 157 mgrm On Sept 25, however, the temperature rose sharply, and a patch of erysipelas appeared about the left ear This spread rapidly, on the morning of Sept 26 a blood-culture showed a growth of *Streptococcus hæmolyticus*, and the value for blood-urea was 265 mgrm On Sept 27 he died

AUTOPSY—Only a partial examination was made The left kidney itself was small, its capsule was thickened and adherent to the surrounding fat On section it presented numerous early abscesses and marked tubular degeneration The left ureter was

FIG 624—Case 1 The bladder has been laid open, and shows the 'valve' over the anterior margin of the vesical outlet



considerably dilated Similar changes affected the right kidney and ureter The bladder was greatly dilated and hypertrophied, although there was little sign of inflammatory change in the mucosa The ureteric orifices were normal

A fine membranous band crossed the internal orifice of the urethra from side to side. Anteriorly its base was attached to the anterior margin of the orifice, its posterior margin was free (*Fig 624*). Microscopically the fold consisted of a mass of œdematous fibrous tissue covered by transitional epithelium.

Case 2 (K H W)—A lad aged 18 years was admitted to the Manchester Royal Infirmary on April 2, 1934. He stated that urination had always been more difficult and more frequent for him than for other boys. Up to the age of 12 years he had had nocturnal incontinence. For five or six weeks before admission his symptoms had been unusually severe, he had to strain very hard to pass urine, and there was marked frequency—every hour or so during both day and night. There was some urgency, but never actual incontinence. Recently he had noticed a tumour in the lower abdomen when the bladder was full, but this disappeared after he had passed urine. Rectal function was not impaired.

ON EXAMINATION—The patient was a slender unhealthy-looking youth. A soft swelling, which disappeared on urination, occupied the hypogastrium and part of the right iliac fossa. The external genitalia were normal, and rectal examination showed that the tone of the anal sphincter was normal. There was no evidence of disease of the nervous system. The urine contained innumerable leucocytes, *Bacillus coli*, and a hæmolytic streptococcus. X-ray examination of the urinary tract did not reveal any abnormal shadows*. On admission the value for blood-urea was 98 mgrm per 100 c.c. He was observed to strain very hard during an act of urination, and passed 240 c.c. of urine with twelve interruptions, which occurred when he relaxed his efforts. Each spurt of urine was of fair force, and appeared slightly more forcible than in cases of complete detrusor paralysis. A No. 15 F rubber catheter was passed, and 500 c.c. of urine withdrawn, without completely emptying the bladder, it would appear that some of this urine must have come from the upper urinary tract, because the bladder was not palpable before catheterization.

Cystogram (Fig 625)—It was surprising that only a small amount of fluid would run by gravity into the bladder. The main cavity of the bladder appeared to be very small, and round it there were numerous diverticula. There was no evidence of relaxation of the vesical orifice, of the so-called 'funnel urethra', or of ureteral reflux.

Bladder-pressure Estimation (Fig 626)—For twenty-four hours the indwelling catheter, which had been inserted on April 13, was removed. During this period the patient passed some urine, and the swelling in the right iliac fossa again became palpable. The pressures were first recorded during emptying of the bladder, and again during the subsequent filling with fluid. It is probable that the curve of emptying pressures was affected by the overflow of urine from the upper urinary tract. The striking feature was the very steep curve of filling, which was quite abnormal, and previously considered (K H W⁴) to be characteristic of a lesion affecting the sacral segments of the spinal cord or the cauda equina. Evidence of powerful detrusor contraction was lacking, and the pressures achieved during the attempts to urinate (shown by the two lines rising vertically from the curve of emptying) appeared to be due to contraction of the abdominal muscles. A

* A spina bifida was present, but in consultation with Mr Geoffrey Jefferson it was decided that it had no significance.



FIG 625—Case 2 Cystogram

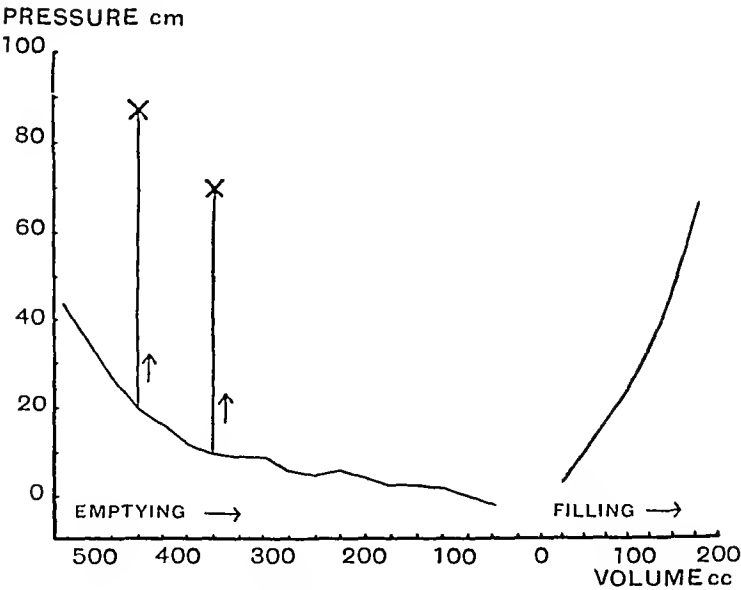


FIG 626—Case 2 Curve showing emptying and filling pressures in bladder

definite sensation of filling was noticed after the introduction of 50 c c of fluid into the bladder

Cystoscopy was carried out under ether anaesthesia. The capacity of the bladder was small. Between huge trabeculae appeared the orifices of numerous diverticula. The right ureteral orifice appeared to be normal, the left was not seen. The trigone was not unduly prominent. Posteriorly the vesical orifice seemed to be normal in outline, but anteriorly and laterally the appearance resembled that of intravesical enlargement of the lateral lobes of the prostate, except that there was no anterior cleft. The posterior urethra could not be seen with the concave sheath. With the convex sheath the posterior urethra appeared to be normal, apart from a slight enlargement of the verumontanum. The prominence which was seen on the anterior aspect of the neck of the bladder did not encroach upon the posterior urethra.

OPERATION—In order to provide suprapubic drainage, on May 5 suprapubic cystostomy was performed under ether anaesthesia. When the interior of the



FIG 627—Case 2. Appearance of valve at operation

bladder was inspected urine was seen to be issuing freely from both ureters. At the base of the bladder was seen a fold of mucous membrane, 1.5 to 2 cm high and of similar breadth (Fig 627). It was 6 to 7 mm in its greatest thickness and soft in consistence. The internal meatus could not be located until a bougie was passed, then it was seen to lie immediately beneath the valvular fold, which appeared to lie naturally over it. The fold was easily and quickly excised, and the bladder was drained by a tube.

For the first three days after operation vomiting and hiccup were present, and the value for blood-urea rose to 304 mgrm per 100 c c. The patient's condition then improved, and by the sixth day the value for blood-urea had fallen to 100 mgrm per 100 c c. On the twelfth day he collapsed very suddenly and died within a few moments.

An autopsy was not obtained. Histologically the fold consisted of loose and vascular fibrous tissue, covered by transitional epithelium.

COMMENT

In both cases, and especially in *Case 2* as a result of the estimations of bladder pressure, it was thought at first that the urinary retention was an expression of neuromuscular dysfunction, which may exist in the absence of somatic neurological signs. In the final analysis it is clear that in both cases only mechanical obstruction was present.

In both cases the position and the structure of the valve-like folds were identical. Their histological appearance does not give any clue to their origin. It has been suggested to one of us (J. R. L.) by Dr. John Beattie, Conservator to the Royal College of Surgeons of England, that they may be a result of the tendency to valve-formation seen in other muscular tubes (e.g., the gut) at levels where epithelium lining different embryological divisions of the tube becomes continuous. In our cases the valves appeared where the allantoic portion of the lower urinary tract becomes continuous with the Wolffian.

The possibility of the presence of this rare condition lends additional emphasis to the rule that a urological investigation, especially in children, should be complete. Only by strict adherence to this rule will cases of obstruction be recognized at a time when sufficient renal function remains to ensure that appropriate surgical treatment will be followed by permanent benefit.

One of the authors (K. H. W.) is indebted to Professor Ramsbottom and Mr. Charles Roberts, of the Manchester Royal Infirmary, for their kind permission to record *Case 2*.

REFERENCES

- ¹ HARRIS, A., "Congenital Vesical Neck Obstruction in a Female Child due to Cup-valve Formation. Open Operation. Complete Recovery", *Amer Jour Surg*, 1933, **xx**, 64.
- ² CAMPBELL, M. F., "Trigonal Curtain Obstruction of the Bladder Outlet", *Jour of Urol*, 1932, **xxvii**, 157.
- ³ WALLACE, W. J., "Unusual Bladder Obstruction", *Jour of Urol*, 1926, **xv**, 325.
- ⁴ WATKINS, K. H., "The Clinical Value of Bladder Pressure Estimations", *Brit Jour Urol*, 1934, **vi**, 104.

AN ANTERIORLY PLACED RENAL PELVIS

By W. K. IRWIN

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THE following case in which the pelvis of the kidney was displaced to the anterior surface instead of being situated at the mesial border seems worthy of record.

HISTORY—Mr. K., aged 37 years, gave a history of pain in the left side of the abdomen extending over a period of three weeks. The pain, which was present all the time, showed itself as a dull ache or in the form of agonizing paroxysms radiating down the line of the left ureter and along the urethra. The patient also complained of urgency and frequency of micturition, nausea, and vomiting.

ON EXAMINATION—There was tenderness in the renal area of the affected side, the urine was free from infection, and X-ray examination failed to produce



FIG 628—Pyelogram showing calices of left kidney directed forwards and medially. Right kidney normal.



FIG 629—Instrumental pyelogram of left or abnormal kidney.

any evidence of urinary calculus. By excretion urography (Fig 628) the pelvis and calices of the right kidney were seen to be normal, while the calices of the left kidney were shown to be directed forwards and medially. Fig 629 is an instrumental pyelogram of the left or abnormal organ.

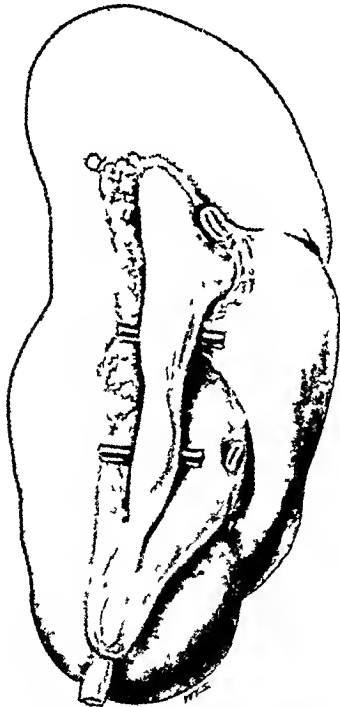


FIG 630—Drawing of excised kidney.

OPERATION (Jan 10, 1934)—Through a lumbar incision I exposed the left kidney, which presented an enlarged anteriorly placed pelvis. Further examination failed to demonstrate anything which might cause obstruction to the pelvic outflow, and a nephrectomy was done. The patient made an uninterrupted recovery and has remained well since the operation.

ANOMALIES OF THE REMOVED KIDNEY—The organ was $5\frac{1}{2}$ in in length, $2\frac{3}{4}$ in in breadth, and $1\frac{1}{4}$ in in thickness. The renal pelvis, as shown in the accompanying drawing (Fig 630), was centrally placed on the anterior surface. As it passed upwards the pelvis separated into two primary divisions—a smaller upper and a larger lower branch. The upper branch separated into two sub-divisions and the lower into three sub-divisions before entering the hilum.

CALCULUS IN AN ECTOPIC KIDNEY

By R. TREVOR JONES

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C. B., aged 16 years, was admitted to Redhill County Hospital, Edgware, for investigation of a recurrent left-sided lower abdominal pain of five years' duration.

ON EXAMINATION—No abnormality was revealed by abdominal examination. The site of the pain was indicated as low down in the left iliac fossa. Dysuria and increased frequency were complained of during the attacks. Urine examination revealed scanty red blood-cells and a few pus cells.

Plain radiograms revealed nothing in the renal areas, but a large round shadow in the sacral region. Cystoscopic examination revealed nothing abnormal, the bladder and ureteric orifices were normal. Uroselectan pyelography revealed a normal right kidney and complete absence of any kidney in the left loin. The



FIG. 631—Skilogram taken three minutes after injection of uroselectan. Absence of any shadow in left renal area.



FIG. 632—Skilogram demonstrating stone in ectopic kidney.

shadow below the promontory of the sacrum was now obscured by the shadow of a hydronephrotic ectopic kidney lying in the hollow of the sacrum. Confirmation of this was obtained by passing an opaque bougie along the left ureter and X-raying it (Figs 631-634).

The urine obtained from the ectopic kidney was infected with streptococci.

OPERATION—The abnormal kidney was removed through a left paramedian incision, retraction of the left rectus muscle, and division of the transversalis fascia, the peritoneum being stripped from the iliac fossa until the aorta and the left common iliac artery were exposed. The ectopic kidney was exposed by tearing through its fatty capsule. The blood-supply of the kidney was by numerous branches from the left and right common iliac arteries, which could be ligated

individually as they entered the upper border of the disc-shaped kidney. The vessels having been ligated, the kidney was withdrawn from the wound and the pelvis and ureter isolated. The ureter was traced to its attachment to the bladder, where it was divided and carbolized.

Convalescence was uneventful.



FIG 633—Pyelogram of ectopic kidney ten minutes after injection of uroselectan



FIG 634—Skingram showing catheter in left ureter

FURTHER REPORT ON A CASE OF TRIGEMINAL NEURALGIA

By J HAMILTON BARCLAY

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IN a previous number of the BRITISH JOURNAL OF SURGERY (1921, ix, No 34, 306) I reported "a case of trigeminal neuralgia in a boy, aged 10 years, treated by intracranial division of the second and third divisions of the nerve"

I now wish to report the subsequent history of this case because I think the after-history is interesting and instructive, and further because I believe that at the time I published the case in 1921 the patient was the youngest authentic case of trigeminal neuralgia recorded (10 years)

The operation was performed at the Hospital for Sick Children, Newcastle-upon-Tyne, on Feb 6, 1920. The second and third divisions of the trigeminal nerve on the right side were divided intracranially. The pain was immediately relieved and remained so for eleven years (till the early part of 1931), when the patient commenced to have twinges of pain in the lower part of the right side of his face. The pains got worse and he came to see me at the Royal Victoria Infirmary, Newcastle-upon-Tyne. There was no doubt the trouble had recurred in the third division of the nerve. I referred him to my colleague, Dr F J

Nattrass, who injected the third division with alcohol in October, 1931. The pain was relieved, but recurred in May, 1933, and the patient again consulted me at the Royal Victoria Infirmary. He was having severe spasms of pain in the distribution of the second and third divisions of the nerve on the right side. With the concurrence of Dr. Nattrass I decided to divide the sensory root of the nerve. The patient was then 26 years of age and married.

OPERATION—This was performed on June 15, 1933. Nembutal, gr $1\frac{1}{2}$, was given by the mouth the night before, and the dose was repeated on the morning of operation, together with morphia, gr $\frac{1}{2}$, and atropine sulphate, gr $\frac{1}{10}$. Open ether was administered and the anæsthetic was assisted by local infiltration with 1 per cent novocain. At operation the old temporal scalp-flap was turned down and the edges of the gap in the skull were exposed. The dura mater was firmly



FIG. 635.—Front and side views of patient's face at present time.

adherent to the edges of the gap and was freed with some difficulty. The dura and brain were lifted up from the middle fossa of the base of the skull. The foramen spinosum, foramen ovale, and foramen rotundum were easily exposed and recognized, and by further elevation of the dura mater the Gasserian ganglion was exposed lying in the cavum Meckelii. The dura was incised and the sensory and motor roots were cut across—as the third division carrying the motor fibres of the nerve had been divided at the first operation it was not considered necessary to differentiate between the motor and sensory roots. The dura and brain were allowed to fall down into position and the flap was sutured, a tissue drain being left in for a couple of days. The eye was covered up with an aseptic dressing to guard against possible corneal ulceration.

The operation was easier than anticipated. Some adhesions and fibrous tissue after the first operation were encountered, but did not constitute a formidable obstacle. There was at times some rather troublesome hæmorrhage, mainly venous,

but this was easily stopped by the coagulating diathermy current and by gauze pressure

AFTER-HISTORY—The day after the operation the patient was very fit and there was complete anaesthesia in the distribution of the trigeminal nerve on the right side. Recovery was smooth and uneventful, he was allowed up at the end of a week, and went home the following day. When last seen on Sept 10, 1934, he was perfectly fit and absolutely free from any pain (*Fig 635*)

HETEROTOPIC BONE FORMATION ASSOCIATED WITH ADENOCARCINOMA IN AN ABDOMINAL SCAR

By ALFRED CLARK

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ALTHOUGH heterotopic bone formation in an abdominal scar is a recognized condition, it is relatively uncommon. The points of interest in this case were the presence of both bone and adenocarcinoma in the abdominal scar.

HISTORY—The patient, a woman aged 51 years, was admitted to the Glasgow Royal Infirmary in August, 1933. At the operation the abdomen was opened by



FIG 636—Photomicrograph of section of mass, showing presence of bone and adenocarcinoma

a right paramedian incision and a large inoperable carcinoma of the pelvic colon was found, the paramedian incision was closed and a colostomy was made. Six months later the patient was readmitted to hospital with a hard mass, about the size of a walnut, situated in the paramedian scar. The mass was excised and, after decalcification, sections were cut. The photomicrograph, *Fig 636*, shows the

presence of bone and adenocarcinoma, and *Fig 637* is a higher-power magnification of a part of the bone

Commentary—The presence of the adenocarcinoma in the scar was probably due to implantation of cells taking place at the first operation, and this itself is rare. According to Leriche and Policard the factors necessary for the formation



FIG 637—A portion of the bone observed under high-power magnification

of bone are an ossifiable medium, an excess of calcium, and an adequate blood-supply, and it is the presence of these requisite factors that permits the development of bone in abdominal scars and other tissues. In this case it is possible that the presence of the adenocarcinoma in the scar accounted for the adequate blood-supply, and thus permitted the development of the bone, on the other hand, the bone formation may have been independent of the adenocarcinoma.

I am indebted to Mr Donald Duff for permission to publish this case

ENDOSTEAL FIBROMA OF THE FIBULA

BY D. F. CAPPELL

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WITH the exception of enchondromata and the exostoses, both of which are commonly multiple and may be familial, simple tumours of the long bones are comparatively rare. The following case, therefore, is worthy of record on account of the rarity of the condition and the consequent difficulty in pre-operative diagnosis.

HISTORY—C. D., aged 32, a dock labourer, was admitted to the Western Infirmary, Glasgow, on Feb. 4, 1928, under the care of Mr. Roy Young, complaining of a swelling in the right leg. In 1917 he had suffered a bullet wound in the right calf, this healed well, and so far as he knew the bones of the leg were not involved. About five or six years before admission he first noticed a swelling in the right calf just above the scar of the old wound. This had increased gradually in size, but had remained painless until two weeks before admission, when it became much larger and painful after a minor injury. The patient's general health was excellent and the family history good.

ON EXAMINATION—A large, firm, non-fluctuant swelling was found in the right calf, which measured 14 in. in circumference as compared with 11 in. on the left side. The growth was fixed to the fibula, but did not involve the tibia. There was no restriction of mobility in the limb. There was no apparent enlargement of glands, the Wassermann reaction was negative and the urine normal. X-ray examination revealed a large tumour mass involving the centre of the shaft of the fibula, the cortex of the bone being expanded, thinned, and at one point ruptured with extension of the tumour mass between the adjacent muscles. The growth appeared to have begun in the medulla and to have subsequently extended through the compact bone. A transverse fracture of the fibula was present (*see Fig. 638*). A few small areas of calcification were noted in the extrafibular tumour, but there was no evidence of bone formation apart from a layer of reactive periosteal bone on the surface of the intramedullary portion.

OPERATION—Although the clinical and radiographical features of the growth did not definitely point to osteogenic sarcoma, it was considered advisable to obtain permission for amputation, but, in view of the doubt as to the malignant nature of the growth, it was decided to carry out microscopic examination of the tumour at the operation. Accordingly, the growth was exposed by Mr. Roy Young and portions from different areas were sectioned immediately and examined by Terry's method (1927). In the portions examined the tumour appeared to be simple in nature, and consequently local excision was performed, the fibula being cut through above and below the growth, which was then separated from the surrounding muscles. A large branch of the posterior tibial nerve was found to pass into the tumour and had to be sacrificed during the removal.

AFTER-HISTORY—This was uneventful. For some time difficulty in walking was experienced, but after three months this had practically disappeared and an excellent functional result had been obtained. The patient last reported on Oct. 2, 1934, and was then found to be in excellent health and able for full work as a dock labourer.

PATHOLOGICAL REPORT—The tumour consists of two main portions, that within the fibula and that external in the surrounding tissues. The naked-eye appearance of the specimen after sectioning in the coronal plane is shown in *Fig 639*. The mass within the fibula measures $7 \times 2.5 \times 2.5$ cm, it fills the interior of the medullary cavity and has produced expansion and thinning of the compact cortex. In most places the tumour mass is covered by a thin shell of dense bone, apparently newly formed during the gradual expansion of the shaft of the fibula. The bossy surface of the growth is shown also by the irregular shape of the expanded bone as seen in the X-ray (*Fig 638*). On the postero-mesial aspect of the fibula

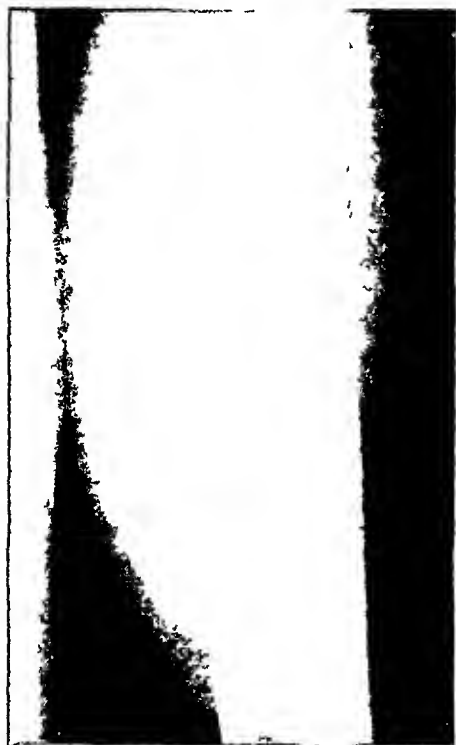


FIG 638—Lateral X-ray photograph. The plates show the expansion of the fibular shaft with the formation of a new layer of periosteal bone on its surface. Scattered foci of calcification are seen in the external tumour medial to the fibular shaft.



FIG 639—Coronal section of the resected fibula and tumour showing the fasciculated appearance of the cut surface with small areas of calcification. Note the expansion of the fibular shaft (x1).

the bony cortex has disappeared and the intramedullary growth is joined to the larger external mass by a pedicle 2 cm in width. The external mass, measuring $16 \times 7 \times 6$ cm, is of more or less ovoid shape, with a fairly smooth bossy surface, and it projects backwards and inwards from the fibular shaft to occupy the calf of the leg. On section the tumour presents an almost uniform glistening whorled appearance resembling white fibrous tissue. The actual tumour tissue appears to be separated from the muscles by a well-defined capsule and is sharply delimited from the medullary cavity and bony cortex, the architecture of which is unaffected except in the immediate vicinity of the growth. A few small irregular nodules of hyaline appearance are seen near the bony attachment, some of these are intensely

calcified, but there is no true bone formation. Areas of cystic degeneration are not present, and the tumour is devoid of areas of pigmentation or hæmorrhage. One half of the growth was preserved as a museum specimen, the other half was thoroughly sectioned, but failed to reveal any areas of more cellular structure than those here shown (*see Figs 640, 641*)

The structure of the growth is not suggestive of neurofibroma, and the inclusion of a large branch of the posterior tibial nerve appears to be incidental. The evidence is in favour of the intramedullary origin of the tumour, and, while the growth arose in the vicinity of the old bullet wound, there is no evidence that it was in any way related to the former injury.

Microscopic examination of numerous portions of the tumour reveals a fairly uniform structure, the growth being composed of whorled bands of elongated spindle-shaped connective-tissue cells separated by an abundant intercellular collagenous stroma (*Fig 640*). At its margin the tumour is well encapsulated,



FIG 640—The greater part of the tumour is composed of rather acellular white fibrous tissue, in which occur a few blood-vessels with thick hyaline walls ($\times 150$)

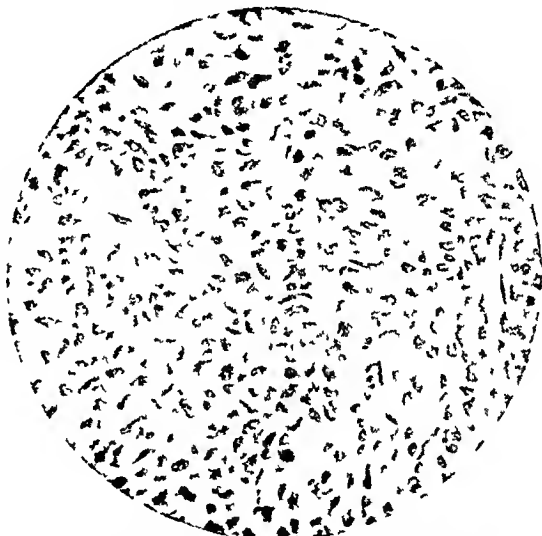


FIG 641—The most cellular area found after extensive microscopic examination of one half of the tumour. The cells are separated by a soft mucoid matrix and are more numerous and

the structure thus being that of a moderately hard fibroma. In other parts the structure is more cellular, the individual elements being stellate rather than fusiform, and the intercellular matrix of looser texture. In a few places the stellate cells are separated by a mucoid matrix, but such myxomatous areas are small and not of frequent occurrence. There is no trace of osseous or osteoid tissue throughout the growth, and the matrix is everywhere of collagenous type. Giant cells of osteoclast type and phagocytes containing altered blood pigment appear to be completely lacking. The structure of the bone above and below the growth appears to be normal, and fibrous transformation of the marrow is quite absent. The blood-vessels generally present thick, rather hyaline walls, but a few thin-walled vessels are found in the more cellular areas (*Fig 641*).

The general structure thus suggests a tumour of simple fibrous nature, or of, at most, a very low grade of malignancy, in view of the favourable outcome of the case, it is proposed to classify the tumour as an endosteal fibroma.

COMMENTARY

The interest of this case lies in the rarity of the condition and in the difficulty of pre-operative diagnosis. The long duration without noteworthy disability, the very gradual increase in the size, and the absence of pain until a minor injury led to pathological fracture, all suggested that the growth was not an osteogenic sarcoma. The site of the tumour in the centre of the fibular shaft is unusual, and the radiographic appearances were not typical of any of the commoner tumours or other lesions of bone.

In the differential diagnosis the localized form of osteitis fibrosa might require consideration. In this disease the onset is usually in early life, and its first appearance at the age of 26 years would be exceptional. Cystic change in the new-formed fibrous mass is the rule in the generalized form of osteitis fibrosa associated with hyperparathyroidism, but it is less common though not unknown in focal osteitis fibrosa (Knaggs, 1926, Emslie, Fraser, et al., 1933). The latter authors record a case of widespread osteitis fibrosa not associated with hyperparathyroidism in which there was fusiform expansion of the left fibular shaft, but in addition changes were present in many other bones. Exploration of the femur revealed dense but highly vascular fibrous tissue at the site of old fractures. Throughout this fibrous mass were small areas of bone without true lamellar structure. In osteitis fibrosa, while the new-formed fibrous mass may distend and destroy the bony shaft, it does not as a rule spread beyond the shaft, and extension in a tumour-like fashion between neighbouring muscles is unlikely. The new-formed fibrous tissue in localized osteitis fibrosa usually contains some osseous or osteoid tissue, and foci of giant cells are met with. The absence of trabecular bone, osteoid tissue, and giant cells in the fibrous growth considered above renders it improbable that the case was one of osteitis fibrosa. Accordingly, we are of the opinion that the case is one of true neoplasm of relatively simple nature arising within the medullary cavity of the fibula.

The growth appears to belong to the group described as medullary fibrosarcoma by Ewing (1928), who states that the prognosis is relatively good and that metastases are unlikely, at least from the less cellular varieties. In the present case there is, after almost seven years, no evidence of local or other recurrence, and the patient, now in perfect health, is able to earn his living as a dock labourer. The good prognosis given from microscopic examination during the operation, and the conservative treatment accordingly adopted, have thus been justified.

In a search of the literature no closely similar case has been found, that most nearly parallel being one of Le Conte (1921), who reported a simple fibroma of the fibula with islets of cartilage in a woman of 21. In this case also the tumour had grown slowly for some years, total removal, which involved sacrifice of the peroneal nerve, was successful, the patient being well at least two years later. In the older literature there are numerous references to fibroplastic tumours of the fibula, but in most instances either these are clearly osteogenic sarcomata, or the details are insufficient for proper identification. Wintrebert and Lardennois (1897) record the case of a man of 42 in whom a large slowly growing tumour developed from the lateral aspect of the fibula at the site of an old injury and spread out into the intermuscular septa, pushing aside the muscles, amputation was performed, but on microscopic examination of the new growth, Bezançon pronounced it to be of

simple nature, being composed superficially of well-formed collagenous fibrous tissue, and in the deeper parts of bony lamellæ and osteoid tissue. Laffan (1893) also recorded a tumour in the head of the left fibula in a man of 52. The growth arose at the site of an injury two years previously, pain was noted in the region four years before operation, and during this period the growth attained a diameter of about three inches. Amputation was performed, and after a stormy convalescence complicated by secondary hæmorrhage, recovery ensued, the patient being apparently well at least seven years after operation. The growth is referred to as a 'sarcoma', but no mention of its structure is made.

None of the above cases is completely parallel to our own, in that the first two showed an admixture of other tissues, e.g., cartilage, osteoid tissue, and bone, while in the third the structure is not described in sufficient detail to make comparison of value. An association with the site of a former injury is noted in the second and third cases as in our own, but the significance of this is not clear.

Accordingly it would appear that simple endosteal fibrous tumours of the fibula and other long bones are exceedingly uncommon. The present account is therefore desirable in order to put on record the clinical and pathological features of such a case, and the satisfactory outcome of local conservative treatment.

SUMMARY

An account is given of a case of endosteal fibroma of the fibula, in which the tumour had burst through the bony cortex and pushed aside the muscles. Partial resection of the fibula together with the extrafibular tumour was performed with satisfactory result, the patient being well and active without recurrence six years later.

I am indebted to Mr Roy Young, Visiting Surgeon to the Western Infirmary Glasgow, for the clinical notes and X-ray plates of the case.

BIBLIOGRAPHY

- ELMSLIE, FRASER, DUNHILL, NIK, HARRIS, and DAUPHINEE, *Brit Jour Surg*, 1933, **xx**, 479.
 EWING, *Neoplastic Diseases*, 1928.
 KNAGGS, *Inflammatory and Toxic Diseases of Bone*, 1926.
 LAFFAN, *Med Press and Circ*, 1893, 499.
 LE CONTE, *Ann of Surg*, 1921, **lxxiv**, 794.
 TERRY, *Jour Pathol and Bacteriol*, 1927, **xxx**, 573.
 WINTREBERT and LARDENNOIS, *Bull de la Soc Anat*, 1897, **lxxii**, 794.

REVIEWS AND NOTICES OF BOOKS

Modern Operative Surgery Edited by G GREY TURNER, M.S., F.R.C.S., F.A.C.S (Hon.), Senior Surgeon, Royal Infirmary, Newcastle-upon-Tyne, etc. Second edition. In two volumes. Medium 8vo. Pp 1764, with 860 illustrations in text and 19 plates. 1934. London. Cassell & Co Ltd. £3 3s net.

THIS work was originally planned and edited by the late Mr Herbert Carson. The first edition was published in 1924. Mr Carson died while arrangements were being made for the production of the second edition, and the editorship was undertaken by Professor Grey Turner.

The general plan of the book has not been altered, though new sections have been added on radium by Mr Geoffrey Keynes and on the surgery of the sympathetic system by Mr Geoffrey Jefferson. The second edition contains 200 more pages and nearly 140 more illustrations than the first, and the extra space has been adequately employed in presenting the improvements in surgical knowledge and technique which the intervening ten years have afforded. The influence of the editor is felt widely throughout the book and is characteristically expressed in an introduction, which compresses the experience of a close observer into a few pages of friendly and broad-minded advice.

One of the chief features of this work is the discussion of the indications for the various operations described, a discussion which includes the choice of anæsthetic and the after-treatment of the patient. The argument as to time and type of operation is perhaps seen at its best in the article on appendicitis.

Limitations of space make it necessary to reduce the descriptions of many operations to a short outline and to omit a discussion of their indications, but the value and scope of the book has been greatly increased in many, though not in all, of the contributions, by the addition to the text of references to the original accounts given by their introducers. The second edition of *Modern Operative Surgery* has fully maintained the tradition of the first, and by amplifying its scope has increased its value to the present generation of surgeons.

Aids to Operative Surgery By CECIL P G WAKLEY, D.Sc. (Lond.), F.R.C.S. (Eng.), F.R.S. (Edin.). Second edition. 6½ x 4 in. Pp 225 + viii, with 3 illustrations. 1934. London. Baillière, Tindall & Cox. 3s 6d net.

WE confess that we took up this little book with the biased feeling that it could not be worthy of much attention, but we are glad to say that a careful perusal at once altered our opinion. It is remarkable how the author has succeeded in including so much of the essentials of his subject within so small a compass and yet how clearly he has described the steps of the operations and their indications. For example, under 'Operation upon the Autonomic Nervous System' the various modern procedures of sympathectomy, ganglionectomy, and ramisection are excellently summarized in four pages. It is a great achievement and should prove very useful for rapid reference.

Applied Anatomy the Construction of the Human Body considered in Relation to its Functions, Diseases and Injuries By GWILYM G DAVIS, M.D., Late Professor of Orthopedic Surgery and Associate Professor of Applied Anatomy in the University of Pennsylvania. Revised by GEORGE P MULLER, M.D., Professor of Clinical Surgery, Graduate School of Medicine, University of Pennsylvania, and five Associates. Ninth edition. Large 8vo. Pp 717 + xii, with 674 illustrations. 1934. London. J. B. Lippincott Company. 42s net.

THIS is certainly one of the most comprehensive and best illustrated text-books on applied anatomy which we know, and we welcome this new and enlarged edition. The number, accuracy, and artistic beauty of the illustrations are alone well worth the purchase of the book.

Its main feature is the close correlation of surgical procedures to the underlying anatomical structures of the body. In the present edition the sections on the abdomen, gynaecology, urology, and the brain and spinal cord have been entirely re-written, whereas those dealing with the limbs remain substantially the same as in former editions. Our only criticism is that the description of the anatomy and surgery of the sympathetic nervous system is very incomplete. We think that the anatomy of the approach to the cervical and abdominal cords and ganglia should be fully described in a work of this importance.

A Synopsis of Surgical Anatomy. By ALEXANDER LEE MCGREGOR, M Ch (Edin), F R C S (Eng), Lecturer on Surgical Anatomy, University of the Witwatersrand, Assistant Surgeon, Johannesburg General Hospital. With a Foreword by Sir HAROLD J STILES, K B E, F R C S (Edin). Second Edition. Crown 8vo. Pp 644 + xx, with 639 illustrations. 1934. Bristol. John Wright & Sons Ltd. 17s 6d net.

WE are not surprised that a second edition of this remarkable and most useful book has appeared so soon. It is packed from first page to last with most useful information and the illustrations are models of diagrammatic clearness. The anatomical basis of surgical procedures is set out in a way which is easily understood and remembered. For example, the anatomy of and many modern operations on the sympathetic nervous system may here be studied in concise and accurate presentation. The section on amputations has been entirely re-written, so as to bring it into line with modern ideas about late results and artificial limbs.

Visceriti e Perivisceriti digestive addominali croniche. By FRANCESCO ZAGARESE. Crown 4to. Pp 359, with 60 illustrations. 1934. Bologna. Nicola Zanichelli. L 40.

IN a preface to this work of his sometime pupil's, Professor Paolucci says that it is something more than an eclectic exposition of the manifold theories and opinions on the subject, it is "a complete and original study, that brings a contribution of the first importance to a difficult problem." He adds, a little naively, that he is particularly grateful because he finds therein all the promptings of his own teaching, all his own opinions, all the diagnostic and therapeutic criteria relied upon in his clinic. No doubt this is true, too, but the author has not confined himself to the work of his own school—indeed, it is very clear that his work in Paris with Pauchet has greatly influenced him—and he has brought a critical and sensible mind to bear on a question that does not leave much room for originality.

It is satisfactory to find that he is not enthusiastic about any mechanical means for dealing with or preventing adhesions. The one clear dictum is this: there is only one thing the surgeon can do for the relief of symptoms due to perivisceritis, and that is to eliminate completely the originating focus of infection. He holds that once this focus (which is easily and often overlooked) is removed, adhesions and the like take care of themselves.

There is a bibliography, mainly Italian and French.

Chirurgie du Pancreas. By P BROCC (Paris) and G MIGINIAC (Toulouse). Large 8vo. Pp 428, with 74 illustrations. 1934. Paris. Masson et Cie. Fr 75.

THIS is a comprehensive and thoroughly up-to-date survey of pancreatic surgery. Of the 423 pages of descriptive text, 114 are allotted to acute pancreatitis, 110 to chronic pancreatitis, and 49 to cancer of the pancreas—the three most important of the pancreatic lesions.

The first section is concerned with the various traumatic conditions of the gland. Owing to its deep and well-protected situation, wounds of the pancreas are recognized only during laparotomy or at autopsy, and many are overlooked owing to insufficient exposure of the gland. Reference is made to the three routes of exposure: (1) through the gastro-hepatic omentum, (2) through the gastro-colic omentum, and (3) by the 'inter-colo-epiploic' route, whereby the transverse colon and its mesocolon are separated from the omentum along the plane of fusion which occurs between them during embryonic life. Subcutaneous injuries—*contusions, lacerations, and ruptures*—are recognizable clinically at one of three periods: (1) immediately, when the associated shock and hæmorrhage necessitate laparotomy, (2) some days after the accident when there has formed an encysted hæmatoma of the lesser peritoneal sac, and (3) some weeks later when a traumatic pseudo-cyst of the lesser peritoneal sac has developed.

The section on *acute pancreatitis*, written by P. Brocq, is based upon 468 collected cases in all of which the diagnosis was verified either at operation or autopsy. A fundamental distinction is made between 'aseptic' cases, where the peritoneal effusion is sterile, and 'septic', the former are the more frequent (386 aseptic to 82 septic). The pathological anatomy and histological changes in the various types of acute pancreatitis are well described and illustrated, and attention is drawn to the frequency with which old-standing histological lesions are met with—sclerosis, arteritis, phlebitis, catarrh of ducts. This indicates that it is on a previously damaged gland that acute pancreatitis has supervened, and is in keeping with the clinical observation that slight attacks often precede the final crisis of acute pancreatitis. The authors have carried out a large number of experiments upon dogs, and their researches and those of other workers have shown that ligation or section of the duct of Wirsung does not produce acute pancreatitis, but that this condition will follow injection into the duct of Wirsung of bile, of duodenal contents, of succus entericus, of microbic and other toxins, and of a 20 per cent solution of calcium chloride. Acute pancreatitis is stated to be primarily an 'auto-digestion' of the gland; the conditions in which activation of the pancreatic ferments is brought about are fully considered and special stress is laid upon the frequent association of acute pancreatitis with disorders of the biliary passages. Early operative treatment is advocated as the only safe course to adopt, and the recent tendency with some surgeons to avoid operation during the period of acute crisis is discountenanced. The essential operative procedures are fully detailed and some useful hints on post-operative management are given. It is estimated that diabetes develops in about 10 per cent of recoveries from acute pancreatitis.

The difficulty of estimating the frequency of *chronic pancreatitis* is noted since it is not characterized by any pathognomonic sign, may remain unrecognized throughout life, and is, moreover, frequently overlooked during the course of abdominal operations. The authors stress the necessity of examining the pancreas in all laparotomies for affections of the stomach, duodenum, and bile-passages. Many troubles that remain unrelieved after cholecystectomy are due to an overlooked associated chronic pancreatitis. There is a full description of the clinical manifestations of chronic pancreatitis and of the laboratory methods of studying the functional state of the gland with regard both to its external and its internal secretions. Compression troubles, while affecting chiefly the biliary tract, may rarely concern the pyloro-duodenal region and produce food-stasis, or by pressure upon the solar plexus may cause attacks of pain which may be so severe as to simulate the gastric crises of tabes. An excellent account is given of the pathological anatomy of chronic pancreatitis and of the associated changes in the biliary passages and adjacent viscera, while a series of fine illustrations of microscopical sections of the various types of chronic pancreatitis adds very materially to the value of the work. Surgical treatment is advocated whenever there is evidence of compression, and the indications for the various operative procedures that may be called for are most judiciously stated.

In the section dealing with *cancer of the pancreas* reference is made to the great difficulty of distinguishing between cancer and chronic pancreatitis whether clinically, or during laparotomy, or at autopsy; histological examination is essential, but a caution is given against lightly undertaking biopsy during laparotomy owing to the risks of post-operative hemorrhage and of auto-digestion of the gland. In no other region can cancer produce such rapid wasting of the body as when situated in the pancreas. The islands of Langerhans usually escape, so that glycosuria is only rarely met with. The symptoms and physical signs receive separate description according to whether cancer affects the head, body, or tail of the gland, and there is a very full discussion of the diagnosis of obstructive jaundice. Cholecyst-gastrostomy is advised for the relief of jaundice, and where this is not feasible, either because of the absence or disease of the gall-bladder or because of the patient's weak general state, the bile should be drained externally either by cholecystostomy or choledochostomy.

Contrary to the opinion of many writers who consider that most *cysts of the pancreas* are of neoplastic origin, it is here stated that of all the fluid swellings of pancreatic origin the most frequent are the 'necrotic' pseudo-cysts arising as a sequel to an acute pancreatitis.

Other special chapters deal with syphilis, tuberculosis, pancreatic calculus, pancreatic fistula, and anomalies of the pancreas.

This volume is a veritable mine of information, and is likely to remain for a long period the standard work of reference in connection with the surgery of the pancreas.

Sincronización quirúrgica Team Standard Operatorio sincronizado Apéndicectomía By Dr GUILLERMO BOSCH ARANA (Buenos Aires) 10½ × 7 in Pp 215, with 212 illustrations 1934 Buenos Aires "El Ateneo"

THIS remarkable book suggests the reduction of a surgical operation—in this case appendectomy—to a rigid formula, which prescribes for each person engaged in its conduct his exact individual movements. All actions are precisely synchronized and are performed without a word spoken. A table of the 'declogue' for each participant—surgeon, first assistant, second assistant, sister, and so on, is set out. A multiplicity of pictures—212 in a book of 215 pages—makes meticulously clear the minutiae of every detail, so that others may emulate the 'team standard' of the author, who is Professor of Surgical Technique in the Faculty of Medicine of Buenos Aires.

Two questions force themselves upon the reader. "Will human nature stand the strain?" and "Is it worth while?" The author's obvious enthusiasm and high ideals might almost persuade the young to answer, "Yes!"

The Management of Fractures, Dislocations, and Sprains By JOHN ALBERT KEY, B.S., M.D., Clinical Professor of Orthopedic Surgery, Washington University School of Medicine, and H. EARLE CONWELL, M.D., F.A.C.S., Orthopedic Surgeon for the Tennessee Coal, Iron and Railroad Company, Birmingham, Ala. Imperial 8vo Pp 1164, with 1165 illustrations London Henry Kimpton 63s net

IN this text-book the authors have given a very comprehensive account of American methods of treatment of fractures, dislocations, and sprains. Part I of the text-book consists of an account of the principles of fracture treatment, and the equipment necessary to carry these out in a hospital of 100 to 200 beds, followed by an account of their complications and the treatment of compound fractures. The end of Part I is occupied by a consideration of medico-legal problems and the Workman's Compensation Law as it affects fracture cases in America. Part II of the text-book is confined to the diagnosis and treatment of specific injuries.

The subject-matter is well arranged and excellently illustrated, but there are many views expressed which would not find general acceptance in this country. One is surprised to read that the authors favour the padded plaster cast, when the unpadded cast—though admittedly more difficult of application—secures immobilization in a way which can never be obtained with a padded plaster. Also spinal anaesthesia is not recommended for reduction of fractures of the lower extremity, although the authors admit they have little experience of it.

In the chapter on first-aid treatment of fractures, the authors, although appreciating the value of a Thomas splint, make the rather surprising statement that they are seldom available, and therefore describe the use of board splints. Also, in fractures of the spine the importance of transporting the patient in the prone position is not recognized and a cumbersome ladder splint is described. In the section on special injuries, fractures of the spine are dealt with in considerable detail. In reference to Kummell's disease, the more generally accepted view that these cases are undetected compression fractures is taken, and a fusion operation is advocated. A considerable amount of space is also devoted to low back sprain and the methods of examination and treatment.

The classification of fractures around the elbow-joint adopted is rather a confusing one, and the authors only recommend the Jones sling as a convalescent dressing, when there is no doubt that many fractures around the elbow-joint can be treated from the very beginning in this way. Also an injustice is done to the teaching of the late Sir Robert Jones in the statement that he recommended treatment in acute flexion, this should be altered to "easy flexion." Many surgeons will also consider eight weeks too early 'to stretch' an elbow-joint, even in the absence of abnormal calcification shadows around the joint.

In the treatment of fractures of the forearm, anterior and posterior, wooden splints are employed and then incorporated in a light plaster cast, a method which must procure very imperfect immobilization and lead to trophic changes in the muscles of the forearm. In the treatment of fractures of the neck of the femur, the advantages of the Smith-Petersen nail are not emphasized although it is agreed that the operation is technically somewhat difficult. The statement that many patients are poor surgical risks leads one to the remark that these patients will tolerate just as badly a long period of fixation in plaster, which gives a poor percentage of union compared with the 'nailing' operation. Fractures of the femur in children

are also better treated by immobilization in a plaster spica, in preference to the older Bryant's method which is described

We think that although this text-book may give an account of fractures and their treatment as practised in a large industrial centre in America, one cannot recommend it to the English reader, because so many of the methods of treatment described are such as would hardly find recognition in a modern fracture clinic in this country

The Treatment of Fractures By LORENZ BÖHLER, Director of the Hospital for Accidents, Vienna, Lecturer on Surgery in the University of Vienna. Translated from the Fourth Enlarged and Revised German Edition by ERNEST W. HAY GROVES, M.S., F.R.C.S., Emeritus Professor of Surgery, University of Bristol. Large 8vo. Pp 578, with 1059 illustrations. 1935. Bristol: John Wright & Sons Ltd. 42s net.

BRITISH and American surgeons will be grateful to Professor Hey Groves for his clear and readable translation of the fourth edition of Lorenz Böhrer's book on the treatment of fractures. Although there is a valuable section on general principles, this work is in no sense a complete text-book, and is obviously not intended for the instruction of the undergraduate student or general practitioner. It is a special monograph presenting in considerable detail both the technique in vogue at the present time and the administrative arrangements of the Vienna Hospital for Accidents, which is now world-famous.

Böhrer's earlier work on fractures of the carpus, fractures of the forearm, and other common types of injury has been fully expounded in previous editions, and his practice in this respect may be said to be stabilized. In the latest edition, which now runs to 578 pages, special consideration is devoted to fractures of the spine (41 pages), fractures of the upper end of the femur (39 pages), and fractures of the os calcis (24 pages). In crush fractures of the spine he has adopted the hyperextension method which is in general use in British clinics and which was many years ago first practised by Davis and others in the United States. In fractures of the femoral neck, without discarding the Whitman method of manipulative reduction and prolonged plaster fixation, Böhrer has become an enthusiastic exponent of the operative fixation of the fracture by means of the Smith-Petersen three-flange pin. It would appear that he now favours the extra-articular operation of Sven Johansson, driving in the nail after the preliminary insertion of two wire guides under X-ray control. Böhrer's special addition to this technique is to use two sets of X-ray apparatus in order to cut down the time necessary to carry out the whole procedure, which, he states, takes not more than fifty to sixty minutes "in lucky cases". He appears to be unduly apprehensive of the dangers of the original method of Smith-Petersen, in which a complete exposure of the fracture is a necessary step, but which, in the hands of the experienced surgeon, takes a comparatively short time.

The attempt to treat severe os calcis fractures on rational mechanical lines is perhaps Böhrer's most notable technical contribution to the treatment of fractures. In this chapter, however, we miss the note of optimism which characterized his earlier efforts to deal with these difficult fractures. Also there is no statement regarding the true end-results in men who are called upon to return to the heaviest type of labour. What we want to know is how many of these individuals can climb ladders and walk about scaffolds.

The salient features of Böhrer's teaching are as follows. The importance of early and accurate reduction, under X-ray control, the value of local anaesthesia, the efficacy of the unpadded plaster cast, the inadvisability of open reduction or fixation of any fractures, except those of the olecranon, patella, and neck of the femur, the importance of avoiding oedema by keeping the limb raised during the early stages of treatment, and by the use of Unna's paste for the lower limb in later stages, the treatment of all wounds by open exposure, without the use of any dressing, the danger of massage and passive movements when applied to recent fractures, the importance of active movements from the very first of all joints not actually fixed, the value of ambulant treatment for many fractures of the leg, especially Pott's, that the bad results in fractures are often due to errors in treatment, rather than to the original injury, the importance of team work, including unity of control and adequate follow-up, and finally the enormous economic value of systematic fracture treatment.

The appendix contains a convincing account of both the clinical and financial efficiency of the fracture organization which Böhrer has established in Vienna. There can be no doubt that this monograph is an essential addition to the library of the fracture service of every large hospital in Great Britain and America.

L'Épaulle Anatomie des Formes extérieures, Anatomie radiographique, Chirurgie opératoire By ANTOINE BASSET, Professeur agrégé à la Faculté de Paris, and JACQUES MIALARET, Interne des Hôpitaux de Paris Royal 8vo Pp 292, with 116 illustrations 1934 Paris Masson et Cie Fr 65

THIS is one of a series of monographs on the joints, that on the knee having appeared already, and that on the elbow being in course of preparation. The anatomical and radiographical sections are very clear and well illustrated. The superposition of the muscles and ligaments over the outline bony skeleton shows many useful points and makes the comprehension of the joint mechanism simple. The radiograms are particularly valuable in giving the normal outline of the bones seen in various positions of the arm, e.g., the arm abducted and the X-ray tube placed in the axilla. Also the X-rays of the shoulder after bismuth injections of the blood-vessels and articular cavity are most instructive.

The chapters on the surgery of the joint are characterized by completeness and by a wealth of illustration. The number and variety of methods for each surgical procedure leave one a little bewildered. Simple methods are hardly touched on, whilst methods of great complexity are described at length. For example, the exposure of the shoulder by splitting the deltoid is much the easiest approach to the joint, also a sling of fascia lata round the neck of the humerus is the ideal method of treating the recurrent dislocation, but neither of these methods finds a place. A great number of ingenious methods of treating the flail shoulder, including different muscle transplants, are described. The book is a very valuable storehouse of information, most useful for reference.

Operationstaktik bei Erkrankungen der Gallenwege By W. M. STERN (Paris) and R. FOURCHE (Nancy) Translated by Dr E. HAYWARD (Berlin) Super royal 8vo Pp 256 + viii, with 203 illustrations 1934 Berlin and Vienna Urban & Schwarzenberg Paper covers, RM 12, bound, RM 13 50

ONE is naturally intrigued by the origin and title of this book. For the work of two French surgeons to be translated and edited by a German is a certain evidence of its worth. Then there is the curious title of "The Tactics of the Operations on the Biliary Passages." By this, we understand from the preface, is meant that not only is technique described, but also the circumstances of the local and general condition which determine the type and extent of each operation. The work is divided into three parts. The first deals with examination and preparation preliminary to operation (10 pages), the second with the details of various operations (206 pages), and the third with associated lesions, complications, and post-operative procedures (36 pages).

The feature of this book which makes it of the greatest possible value is the number and character of the illustrations. These are nearly all about three-quarter-life-size line drawings which, whilst of great artistic merit, are wonderfully clear and diagrammatic.

On Osteogenic Sarcoma By JACOB VAN DER SPEK Royal 8vo Pp 217, with 24 X-ray plates 1933 Kemink en Zoon N.V., Overden Dom, Utrecht

THIS is an excellent monograph in English on bone sarcoma in which a very good review of modern pathology, prognosis, and treatment is given. The chief original material is derived from forty-two cases observed in Noordenbos' clinic at Amsterdam. The chief value of the book consists in the fair and complete summary of modern views, both American and Continental, on the subject. The dangers and fallacies of biopsy as a method of diagnosis are fairly pointed out. These tumours are often of such varied structure that the examination of a small piece is of little value, so that in reality the whole tumour should be excised and sectioned before its nature can be known accurately. A just protest is made against the attitude of non-intervention in most cases. Although the proportion of five-year cures may be small, yet the prognosis is certainly better if radical amputation or disarticulation is done than if the case is left alone, and, moreover, there is diminution of pain and suffering. The last section of the book deals with giant-celled sarcoma. It is clearly proved that the operation of enucleation is unsatisfactory and should be abandoned for resection.

Head Injuries By L. BATHIE RAWLING, M.B., M.Ch. (Cantab.), F.R.C.S., Consulting Surgeon to St Bartholomew's Hospital and to the West End Hospital for Nervous Diseases. Demy 8vo. Pp. 86 + vi, with 22 illustrations. 1934. London: Oxford University Press. 7s. 6d. net.

In his preface to this book the author warns his readers that he intends to treat this highly important subject in a personal and dogmatic manner. In many ways this has produced an admirable result, and the opinions of an authority such as Mr. Bathie Rawling are necessarily weighty. The book seems rather too elementary, and the author slurs over some points on which we would have valued his opinions. Thus the indications for decompression in cases of cerebral irritation are summarily dealt with. It is on such a point that we should like to know Mr. Rawling's procedure. The treatment of persistent cerebrospinal fluid loss is also omitted.

The book gives a clear and easily readable account of the commoner skull fractures and the usual forms of cerebral damage. It is clearly paragraphed and reference is easy. We are in complete agreement with the author in condemning the routine administration of hypertonic intravenous salines in head injuries. Such exhibition should always be controlled by lumbar puncture and the clinical picture. The author rather deprecates the use of the spinal manometer as an adjunct to lumbar puncture. He tells us that frequent experience enables one to estimate the pressure. We feel that this is retrograde. Mistakes will occur while this degree of experience is being obtained. Loss of fluid while the surgeon estimates the pressure may be harmful. An astute clinician can estimate the blood-pressure by a finger on the pulse or the number of red cells by examination of the mucous membranes, but few would now dispense with the sphygmomanometer or the blood-count.

The need for frequent re-examination of the clinical and neurological state in the management of such cases could be more stressed, as changes are notoriously rapid. These criticisms are, however, minor, and the book will be read with profit by all who are called upon to treat cases of head injuries.

Diseases of Children Edited by HUGH THURSFIELD, D.M. (Oxon), M.A., F.R.C.P., Physician, Hospital for Sick Children, Great Ormond Street, Consulting Physician, St Bartholomew's Hospital, and DONALD PATERSON, M.D. (Edin.), F.R.C.P., Physician to Out-patients, Hospital for Sick Children, Great Ormond Street, Physician in Charge of Diseases of Children, Westminster Hospital. With contributions by 36 authors. Third edition. Royal 8vo. Pp. 1152 + vii, with 277 illustrations. 1934. London: Edward Arnold & Co. 50s. net.

THE third edition of this standard work, whilst retaining its general arrangement and outlook, contains much that is new and revised, both in illustrations and text. New articles on blood transfusion, orthodontic treatment, diseases of the accessory nasal sinuses and the ear, and on cystoscopy and pyclography, have added breadth to the surgical aspects of the volume. In general, however, the book does not attempt to go deeply into the surgical problems of childhood, but rather to fit them into their appropriate place with a much wider medical background. Surprisingly, congenital pyloric stenosis has been delegated to a surgical contributor. Without criticism of the substance of the article, which is excellent, we doubt the wisdom of this, the operation being an incident only in a disease with pre- and post-operative periods complicated by problems essentially medical. The writer, we think, does not emphasize sufficiently the importance of early diagnosis as being the factor responsible for the lower mortality in private patients.

As one of the major emergencies in childhood acute osteomyelitis might well receive more attention and space to bring it into a proper perspective in a text-book essentially for the practitioner, yet one in which so much is devoted to the rarer forms of bone disease.

In spite of such minor criticisms, which are easy in a volume covering such a wide field, we feel the book is a most valuable compilation which will be widely read and appreciated.

Report on Seventh International Congress of Military Medicine and Pharmacy and Meetings of the Permanent Committee (Madrid, Spain, May 29-June 3, 1933). By CAPT. WILLIAM SEAMAN BAINBRIDGE, M.C.-F., U.S.N.R., Member of the Permanent Committee, Delegate from the United States. Large 8vo. Pp. 88. Menasha, Wis.: George Banta Publishing Co.

CAPTAIN BAINBRIDGE has again performed a valuable service in reducing to readable bulk the voluminous report of this International Congress. The subjects dealt with were as follows:

(1) General principles regarding medical services in war time their application to the new rulings of the Geneva Convention (2) Preventive vaccination in the Army, Navy and Air Force (3) Treatment in advanced posts of urgent surgical caseload in a war of movement Scheme of a specialized formation its technical organization and its employment from a tactical viewpoint (4) Preserved foods as a regular ration for soldier in peace time or in the field their modes of preparation and analysis (5) Comparative study of odonto-stomatological and administrative services in the different Armies, Navies and Air Forces (6) (Veterinary Section) Glanders (a) Clinical and prophylactic study, (b) Bacteriological study, (c) Anatomohistopathological study

There is a short foreword by Rear Admiral P. S. Rossiter of the United States Navy. The little book is well printed and produced. Some errors in the names of the delegates are noted, accounted for by mistakes in the original proceedings.

Benign Encapsulated Tumours in the Lateral Ventricles of the Brain. Diagnosis and Treatment. By WALTER E. DANDY, M.D., Adjunct Professor of Surgery, Johns Hopkins University. Large 8vo. Pp. 189. viii with 83 illustrations. 1934. London: Baillière, Tindall & Cox. 20s. net.

This monograph is the outcome of an analysis of 15 cases of primary benign encapsulated tumours in the lateral ventricles of the brain. All were found at operation, and 14 totally removed. The mortality rate was low, only 3 deaths having occurred in the whole series. In addition to his own 15 cases, Dandy has been able to collect 25 cases from the literature, of which 23 were discovered at autopsy.

The importance of ventriculography is stressed at great length, and Dandy considers that when used correctly, such an investigation is without risk to life or function. It may be said that the only satisfactory treatment of these tumours is complete removal. The operative risk is reduced by the standard use of the electro-cautery, which has done so much to make all types of cerebral surgery safe. The other factors which matter are the use of vertin anaesthesia and continuous suction of the wound area.

The monograph, like all the works of Dandy, is a record of fine and critical observation and judgement, combined with superb operative technique. The illustrations are excellent and could not be bettered, while the analytical tables embodying every case will be found most useful.

Biological Flaps. By J. F. S. ESSER. Published by the Editor of the *Institut Esser de Chirurgie Structure*, Monaco.

ANYTHING from the pen of J. F. S. Esser must be most carefully studied by any reconstructive surgeon. When his epithelial inlay was first published in English in March, 1917, few of the surgeons who read it were alive to the immense possibilities that lay behind the thought. Similarly, to a modified extent, a certain type of antagonism was felt towards his rotation flap. The truth about these two procedures as practised by English-speaking races is that the ideas of both are eminently sound and when suitably modified of great practical value. It would seem that 'the ideal biological' outlook, as conceived by the inventor, has overshadowed in his mind the meticulous refinement that is demanded of the plastic surgeon in the United States or British Empire. Thus also does this new work, devoted to the subject of artery flaps, first strike a note of impracticability, but on reading deeper and taking it in conjunction with the transplantation of fingers and toes and the possibility of even larger limb replacements, one is forced to the conclusion that Dr. Esser has again struck a principle absolutely sound from the biologic point of view. The fact that many of the illustrated cases are poorly finished, or that they give evidence that similar results could be obtained by flaps not specially arterial in design, should not blind the reader to the immense value of this original contribution.

Take, for instance, the simple case of the war injury labelled 6D 39, 6D 40. The same flap could have been utilized with an ordinary pedicle based on the temporal artery, the pedicle returned to the scalp with no more scarring than exhibited in this case, and without the joining scar running down the patient's cheek which is necessary to embed the artery part of the pedicle in Esser's method. That the writer cannot detect any superior advantages of this

method over the established ones of the present day for the kind of injuries depicted in the book does not detract from the great value of the contribution or from its possibilities. The record of the work done as depicted in the book is indicative of the immense labour and ingenuity of the surgeon, and provides a monument of gratitude to him for the success with which these war wrecks have been converted into human beings.

The book is beautifully printed, and, considering the difficulties of obtaining records during the War, the illustrations are both profuse and clear, and there is no suspicion of the slightest dishonesty in the method of taking the photographs. One of the best restorations illustrated is that of L 1 to 5, but why he should be introduced twice complete within a few pages is not quite apparent. One of the most important contributions in the book is the stress which Esser places on the value of the venous and lymphatic supply as compared with the arterial. It has long been recognized that a flap dies through failure of the venous return rather than that of the arterial supply.

It may well be that the development of this biological artery flap, when applied to restorations in which the donor site is on the body, will lead to a great simplification of these methods, and a great saving of time and money. Most particularly is one impressed with the need for experimentation to solve the question of cross-grafting from one patient to another, or even from a corpse. This problem would appear to be linked up with the gradual immunization of the tissues of the recipient to those of the donor. It is to be noted that the book is published by the Editor of the Institute Esser, which is an organization backed by the most scientific and influential people, having as its chief aim a non-political and international centre for reconstruction surgery. The establishment of such an institute will be an amazing tribute to the energy and imagination of Esser.

X-ray Interpretation By H. CECIL H. BULL, M.A., M.B., M.R.C.P., Physician and Hon. Radiologist, Royal Waterloo Hospital, London. Demy 8vo. Pp. 382. 110s., with 280 illustrations. 1935. London: Oxford University Press. 21s. net.

THE author has attempted to compress into some 370 pages nearly the whole of X-ray diagnosis, and a good deal of X-ray anatomy. Many of the simpler conditions are dealt with at length, such as fractures, so that the more difficult and complicated investigations are treated somewhat briefly. The chief value of the book, therefore, would appear to be for those requiring an introduction to radiology. The illustrations are all simple line drawings and silhouettes, and no reproductions of radiographs are shown. In such a section as that on the stomach this is no great disadvantage, but in lesions of the chest or bone diseases it is very difficult to understand the line drawings, unless one is already very familiar with the ordinary X-ray appearances.

The 1934 Year Book of Radiology Edited by CHARLES A. WATERS, M.D. (Diagnosis), and IRA I. KAPLAN, B.Sc., M.D. (Therapeutics). Large 8vo. Pp. 512, with 454 illustrations. 1934. London: H. K. Lewis & Co. Ltd. 19s. net.

THIS book follows on the lines of the past volumes and gives an abstract (with references) of most of the important articles published in the past year. The numerous reproductions from the radiograms are clear, and the present volume, together with its predecessors, forms a valuable reference work.

Selecting a few of the abstracted articles of surgical interest, one might mention those on arteriography, an article describing some new experimental work on the intervertebral discs, and an investigation of the emptying of the gall-bladder after cholecystography. On the X-ray technique side mention is made of the demonstration of enlarged adenoids, and a vertical projection for the neck of the femur which may have considerable bearing on the treatment of fractures in this region. Many cases of bone diseases and tumours are quoted and a case of osteitis fibrosa cystica with a tendency to spontaneous cure. The X-ray examination of the colon in a case of Hirschsprung's disease before and after sympathectomy is also of interest.

Judging from the abstracts, more and more importance is being attributed to the pre-operative irradiation of malignant tumours, particular mention being made of breast cancer.

and renal tumours. In this connection it is emphasized that wound-healing proceeds normally after properly applied radiation. Various auxiliary methods of therapy in cancer are discussed, and the opinion is expressed that the beneficial effects of colloidal selenide therapy are chiefly due to the radiation used with it. Chronic inflammatory conditions are often benefited by radiation, of recent advances, the immediate relief and cure of post-operative pyrosis by radiation should be useful to surgeons, but few would care to use it as the main measure in gas gangrene.

There is a full index of subjects and authors to facilitate quick reference to the various subjects. A perusal of this book should prove of use to the surgeon as well as the radiologist.

Fuss und Bein ihre Erkrankungen und deren Behandlung By Prof Dr med GEORG HOHMANN (Frankfurt a M.) Second edition Royal 8vo Pp 380 + 8, with 326 illustrations 1934 Munich J F Bergmann Paper covers, RM 24, bound, RM 25 80

THIS book was originally written in 1923 and dedicated to Professor Fritz Lange on his seventieth birthday. It is a carefully written and well-illustrated book dealing with the anatomy, physiology and pathology of the foot, especially in regard to various deformities.

The section on gymnastics and physical treatment is very interesting as giving a well-ordered plan for massage and exercises applied for deformed feet. There is an elaborate account of various arch supports without very adequate discussion as to their rationale. In the treatment of various types of talipes and flat-foot great prominence is given to cuneiform osteotomies of the os calcis, navicular, and astragalus. In the treatment of hallux valgus, too, there are a great number and variety of operations described and discussed, chiefly cuneiform osteotomies and muscle transplants. We must admit to being a little bewildered by the complexity of these suggestions.

The so-called arthrosis and arthritis deformans of all the joints of the lower limb is figured and discussed. Many of the minor ailments, e.g., corns, ingrowing toe-nails, and varicose veins are included in this monograph, which certainly contains a great wealth of material and much subject for reflection and discussion.

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

- Operative Surgery** By C R WHITTAKER, F R C S (Edin), F R S E Fifth edition Crown 8vo Pp 228, with 53 illustrations 1934 Edinburgh E & S Livingstone 4s 6d net
- A Short Practice of Surgery** By HAMILTON BAILEY, F R C S, Surgeon, Royal Northern Hospital, etc, and R J MCNILL LOVR, M S (Lond), F R C S, Surgeon, Royal Northern and Metropolitan Hospitals, etc Second edition Demy 8vo Pp, 988 + viii, with 731 illustrations 1935 London H K Lewis & Co Ltd 30s net
- Surgical Errors and Safeguards** By MAX THOREK, M D, Professor of Clinical Surgery, Cook County Graduate School of Medicine, etc Second edition Large 8vo Pp 696 + xv, with 668 illustrations 1935 London J B Lippincott Co 45s net
- Clinical Pathology of the Jaws with a Histologic and Roentgen Study of Practical Cases** By KURT D THOMA, D M D, CHARLES A BRACKETT, Professor of Oral Pathology in Harvard University, etc Large 8vo Pp 643 + viii, with 423 illustrations 1934 London Bailliere, Tindall & Cox 38s net
- Handbook of Anaesthetics** By J STUART ROSS, M B, Ch B, F R C S E, Late Lecturer in Practical Anaesthetics, University of Edinburgh, and H P FAIRLIE, M D, Anaesthetist to the Western Infirmary, the Royal Hospital for Sick Children, and the Dental Hospital, Glasgow Crown 8vo Pp 299 + xv, with 66 illustrations 1935 Edinburgh E & S Livingstone 10s 6d net
- Aids to Surgery** By CECIL A JOLL, M S (Lond), M D (Bris), B Sc (Lond), F R C S, Senior Surgeon and Lecturer on Surgery to the Royal Free Hospital, etc, and REGINALD C B LEDLIE, M B, B S (Lond), F R C S, Surgeon to the Miller General Hospital, etc Sixth edition Fcap 8vo Pp 612 + v, with 44 illustrations 1935 London Bailliere, Tindall & Cox 7s 6d net
- Collected Papers of St Mark's Hospital, London including a History of the Hospital Centenary Volume, 1835-1935** Compiled by the Medical Committee Crown 4to Pp 440 + xvi, with 96 illustrations 1935 London H K Lewis & Co Ltd 30s net
- Surgical Diseases of the Chest** By EVARTS AMBROSE GRAHAM, A B, M D, F A C S, Professor of Surgery, Washington University School of Medicine, St Louis, JACOB JESSE SINGER, M D, F A C P, Associate Professor of Clinical Medicine, Washington University School of Medicine, St Louis, and HARRY C BALLON, M D, C M, F A C S, formerly Assistant Professor of Surgery, Washington University School of Medicine, St Louis Large 8vo Pp 1070, with 637 illustrations 1935 London Henry Kimpton 65s net
- Vortage aus der praktischen Chirurgie** Edited by Prof Dr ERICH LEXER, Direktor der Chirurgischen Universitäts-Klinik, München Part I *Die pyogene Allgemeinfektion und ihre Behandlung*, by Prof Dr ERICH LEXER Royal 8vo Pp 31 1935 Stuttgart Ferdinand Enke RM 1 60

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ATLAS OF PATHOLOGICAL ANATOMY

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FASCICULUS X
GANGRENE FIBROCYSTIC DISEASE OF BONE
MISCELLANEOUS CONDITIONS
Compiled by E K MARTIN, M S, F R C S

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LXXII GANGRENE

GANGRENE is a term of clinical significance used to denote death of anatomically recognizable parts of the body.

The sequence of pathological phenomena which ends in gangrene is similar in plan to that which leads to necration and sloughing and in general terms is brought about by interference with the circulation through a part and by damage to the tissues concerned through physical trauma or toxic agents. Among the latter bacterial toxins predominate.

The clinical varieties of gangrene are dry and moist, of which dry gangrene is the result of reduction in the local arterial blood-supply while moist gangrene is mainly of infective origin.

Dry Gangrene—The most typical form of this variety is senile gangrene. This affects the lower limb and usually commences in the great toe. The patient is an elderly person in whom the circulation through the lower limb has been gradually diminished by reduction in calibre of the arteries. Owing to disease of the arterial walls the adaptability of the circulation through the limb is diminished and the final onset of gangrene is caused either by arterial thrombosis or by the occurrence of a minor degree of superficial inflammation to which the rigidly determined circulation cannot be adapted.

Gangrene appears first as a dusky discoloration of one or more toes or as a blister which darkens in colour. The skin of the affected toe becomes translucent greenish and finally black through the production of sulphide of iron from the hæmoglobin of the extravasated blood. The whole toe shrinks and its surface becomes dry through evaporation of its fluid contents. The gangrene extends up the limb to a level at which the circulation collateral to the thrombosed artery is able to maintain the vitality of the tissues. At this point a line of demarcation is formed. It begins as a narrow red line which is the first stage of the process by which the still living tissues attempt to cast off the gangrenous part. This process is an inflammatory reaction of the living tissues which ulcerate where they are in contact with the dead part. The ulcer gradually extends through the soft parts down to the bone and since the blood-supply of the deeper tissues is more abundant than that of the skin the granulating surface of the line of demarcation shelves distally towards the bone. Where the process continues without interruption the resultant stump is conical. Owing to the progressive nature of the arterial disease and of the tendency to thrombosis, the gangrene often extends up the limb again above the original line of demarcation until a similar reaction is repeated at a higher level.

The onset and progress of dry gangrene is usually accompanied by pain, which is often severe, but owing to the absence of absorption from the dead part, constitutional symptoms are slight.

The progressive vascular occlusion which may end in gangrene is characteristic of senile atheroma and arteriosclerosis and of the corresponding vascular changes seen in diabetes, sclerosis and calcification of the media, syphilis, and thrombo-angitis obliterans.

Dry gangrene also occurs in frost-bite and in Raynaud's disease when the spasm of the arterial wall is long continued. In Raynaud's disease the fingers are affected more often than the toes, and the gangrene may be bilateral.

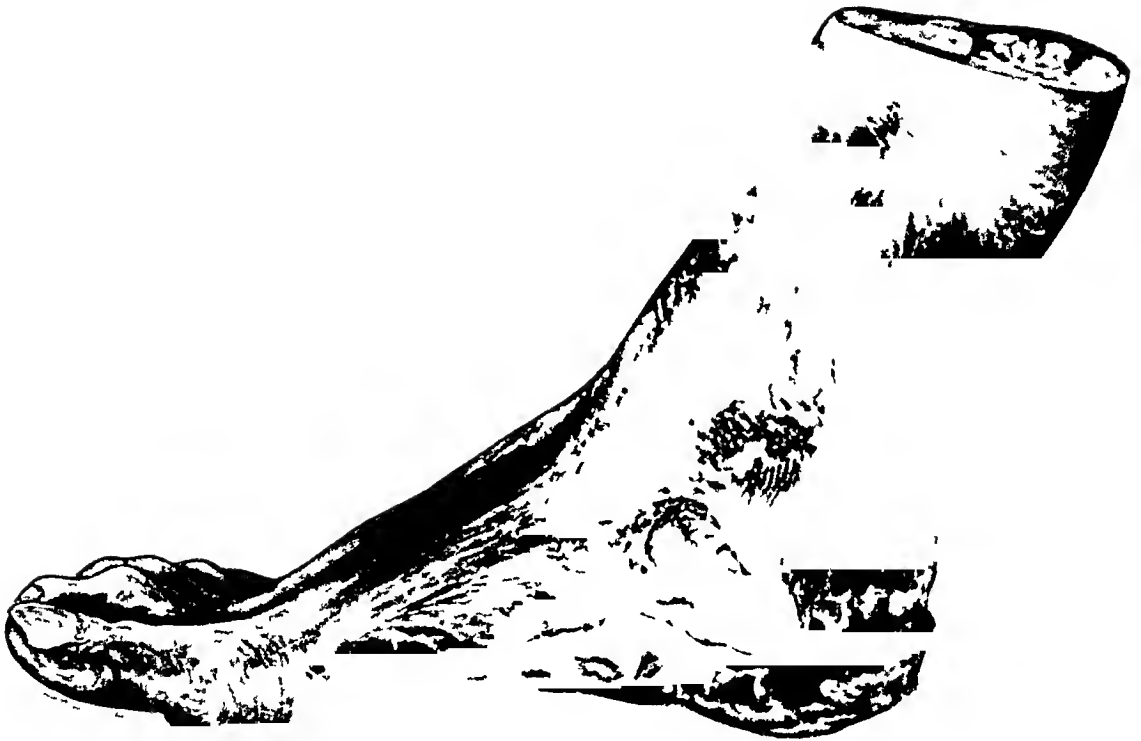
Ligation of the main artery of a limb is seldom followed by gangrene if the artery is a healthy one.

Carbolic Gangrene is a variety of dry gangrene caused by the application of a carbolic compress to a finger. The carbolic need not be strong—1 per cent is sufficient—and the patient feels no pain owing to the analgesic effect of the solution. The gangrene is partly due to the locally destructive action of carbolic on the tissues, and partly to a similar action on the vessels leading to thrombosis.

Moist Gangrene is characterized by retention of fluid in the dead part and the consequent growth of saprophytic microorganisms, particularly those of the anaerobic group. This occurs in characteristic form when the main artery and vein are occluded simultaneously as the result of a lacerated wound through which infection is introduced. The limb swells rapidly owing to inflammatory exudation from the vessels of the collateral circulation, and the swelling progresses until the relatively inelastic fascial envelope of the limb is stretched to its fullest extent. When this point is reached the circulation is arrested and the limb becomes cold. The rapidly increasing edema is accompanied by change of colour to post-mortem type, except in the case of gas gangrene, where the characteristic brown coloration of the skin is seen. Blisters containing foul-smelling fluid may appear on the skin.

Moist gangrene is characterized by rapidity of spread, by the absence of a line of demarcation, and by prolonged constitutional disturbance.

DRY GANGRENE
(ARTERIAL LIGATURE)



A right foot and leg removed three weeks after ligation of the main
vessels on the proximal side of a popliteal aneurysm
Museum of the Cancer Hospital, London, 1226

ANEURYSM OF POPLITEAL ARTERY

A right popliteal artery on which a saccular aneurysm has developed
The lumen of the sac is occupied by blood-clot

Museum of the Cancer Hospital London, 1227

Microscopic Structure There is localized thickening and fibrosis of the intima with destruction of the internal elastic lamina and chronic inflammatory infiltration of the media

Clinical History -The patient was a man aged 53 who had noticed a swelling at the back of the right knee for six months. He had suffered intermittent throbbing pain which radiated down the calf and for three weeks he had been unable to bear weight on the limb.

On examination there was a soft pulsating tender swelling in the right popliteal fossa. The knee was held in flexion and there was pain on passive movement. There was some wasting of the muscles of the calf. The Wassermann reaction was strongly positive.

There was ankylosis of the left knee as a sequel to operation for injury to a semilunar cartilage.

The aneurysm was treated by ligation of the main artery and vein immediately to the proximal side of the sac. Two days later there was severe pain in the calf and the foot became cold and numb. The skin became discoloured and at the end of a week there was dry gangrene of the toes. Two weeks later the gangrene had extended to include the lower third of the leg. The limb was then amputated through the lower third of the thigh.

ANEURYSM OF POPLITEAL ARTERY



MUSEUM OF THE CANCER HOSPITAL, LONDON 1227

NO 37—SUPPLEMENT

SENILE GANGRENE

A right foot with part of the leg

The right great toe is dry black, and shrivelled and is separated from the rest of the foot by a broad ulcerated line of demarcation. The neighbouring skin is red and with the subcutaneous tissue is thickened by œdema. The lower two-thirds of the leg is œdematous and of a brownish colour.

Museum of University College Hospital 112

CLINICAL HISTORY—The patient was a man, aged 67, who had had typhoid fever in the South African War with a left femoral thrombosis. Eight months before admission to hospital he began to have pain in both feet, and two months later a blister appeared on the right great toe. This was followed by gangrene of the toe. There was no change in the other foot. The gangrenous toe was treated by fomentations for six months, after which the patient came to hospital.

On examination the right great toe was gangrenous up to the base of the metatarsal and was separated from the rest of the foot by a deeply ulcerated zone of demarcation. There was little pain. Both legs were œdematous and discoloured almost to the knee. The second right toe was brown and shrivelled. No pulse could be felt in the dorsalis pedis or posterior tibial arteries on either side and the right femoral artery was thickened. Urine normal.

X-ray examination of the right leg showed calcification in the posterior tibial artery and irregular decalcification of the bones of the foot.

Amputation in the lower third of the thigh was followed by satisfactory healing of the flaps. The femoral artery was thickened and obliterated by organized clot at the point of division.

SENILE GANGRENE



GANGRENE FOLLOWING VASCULAR OBSTRUCTION

A left foot amputated for dry gangrene

The foot is of a violet and reddish-brown colour which is most pronounced in the sole. On the dorsum the discoloration is less intense and the limit of gangrene is sharply defined. It is marked in places by early ulceration. The toes are shrunken and the skin is wrinkled.

On the cut surface of the section of the leg the anterior tibial artery is pervious. The walls of the posterior tibial artery are thickened and calcified and its lumen is completely occluded.

Hunterian Museum R.C.S. 2371

MICROSCOPIC STRUCTURE —The posterior tibial artery shows extensive calcification of the media and thickening of the intima.

CLINICAL HISTORY —The patient was a man aged 70.

GANGRENE FOLLOWING VASCULAR OBSTRUCTION



GANGRENE FOLLOWING ARTERIAL DISEASE

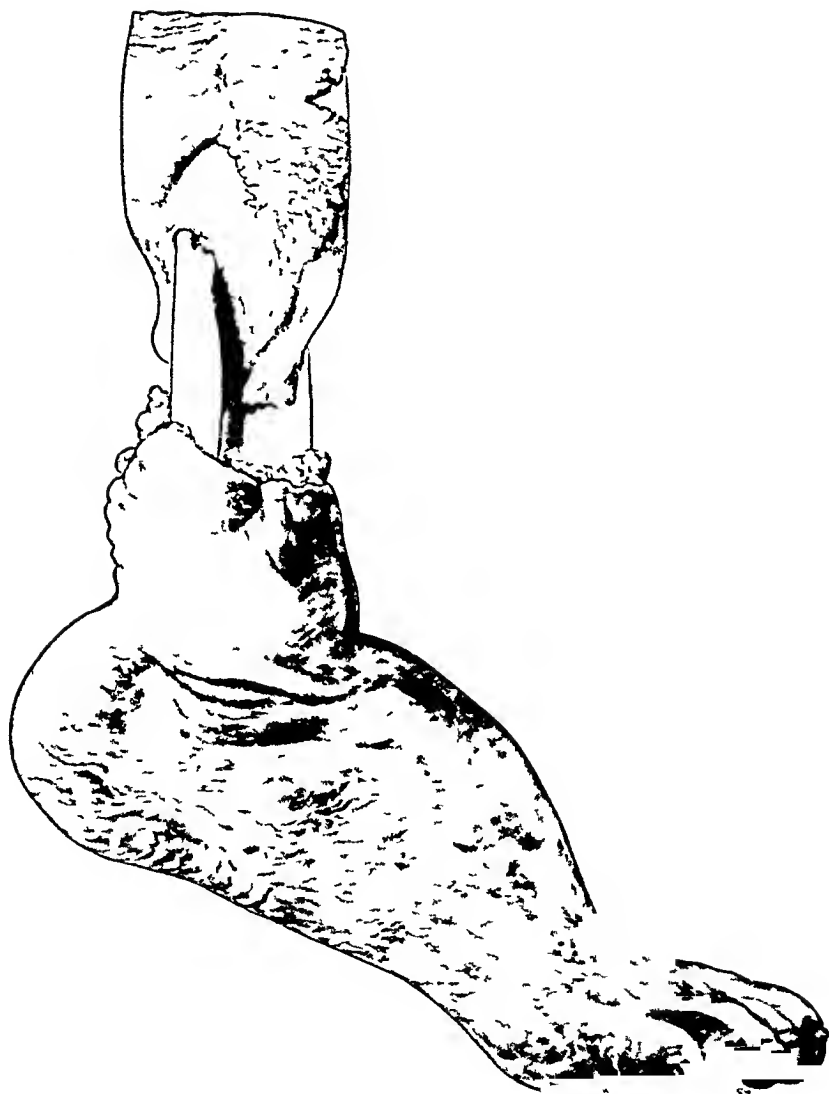
A foot with the adjoining part of the leg

The foot is dry, shrivelled, and black. In the lower part of the leg the soft parts have undergone spontaneous separation, and an interval of 1 in separates the living from the dead tissues. The exposed portions of the tibia and fibula are bare and necrosed, and in the immediate neighbourhood of the living tissues are in process of detachment. There is a deep ulcerated groove round the tibia, and the solution of continuity of the fibula is complete. The residual stump which was in process of formation is long and conical.

Hunterian Museum R.C.S. 2391

CRITICAL HISTORY.—Removed by amputation

GANGRENE FOLLOWING ARTERIAL DISEASE



DRY GANGRENE OF PENIS

A penis divided by longitudinal section

The anterior part of the penis is in a condition of dry gangrene and is almost black

The section shows a stone impacted in the fossa navicularis

Pathological Museum University of Sheffield,

MICROSCOPIC STRUCTURE —Some arterial thickening No endarteritis obliterans

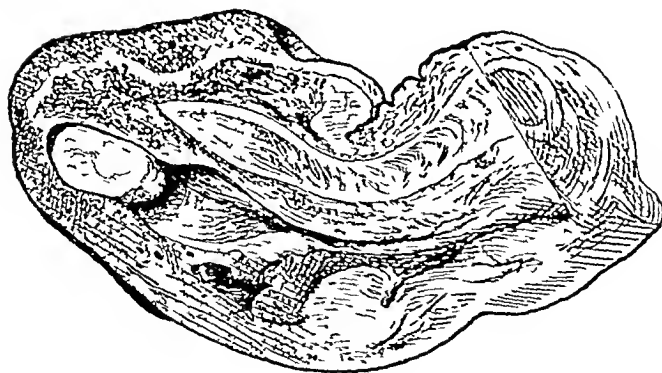
CLINICAL HISTORY —The patient was a man, aged 66 who was admitted to hospital suffering from retention of urine. He was living in a lodging house in the city, was completely deaf, and had no relations so that it was not possible to get an adequate history but his landlord stated that there had been blood on the bedclothes for the past week.

On examination the bladder was distended up to the umbilicus and the distal part of the penis was gangrenous. The gangrenous part was black and dry but some serous fluid exuded from the glans on pressure.

At operation, the bladder was drained suprapubically and the penis was amputated a few days later. The patient died two days after operation without regaining consciousness.

On section of the penis after removal a stone was found impacted in the fossa navicularis and it was thought that the patient had caused a paraphimosis in his attempts to remove it.

DRY GANGRENE OF PENIS



GANGRENE FOLLOWING THROMBOSIS

Parts of two gangrenous hands

In the left hand suppuration of the gangrenous part has occurred at the carpo-metacarpal and in the right hand at the metacarpophalangeal articulation

On both sides the gangrene is dry and the tissues are little changed beyond being shrunken and discoloured. The nails have remained adherent

Hunterian Museum R.C.S., 2341

CLINICAL HISTORY.—The patient was a woman of feeble constitution aged 36 who had been subject to asthma and valvular disease of the heart for sixteen years. During convalescence from typhoid fever the tip of her nose and the ends of both fingers and toes became gangrenous. The nose and toes recovered with slight loss of substance but the parts of the hands illustrated separated spontaneously with good recovery. Both radial and ulnar arteries pulsated feebly on both sides.

GANGRENE FOLLOWING THROMBOSIS



MOIST GANGRENE

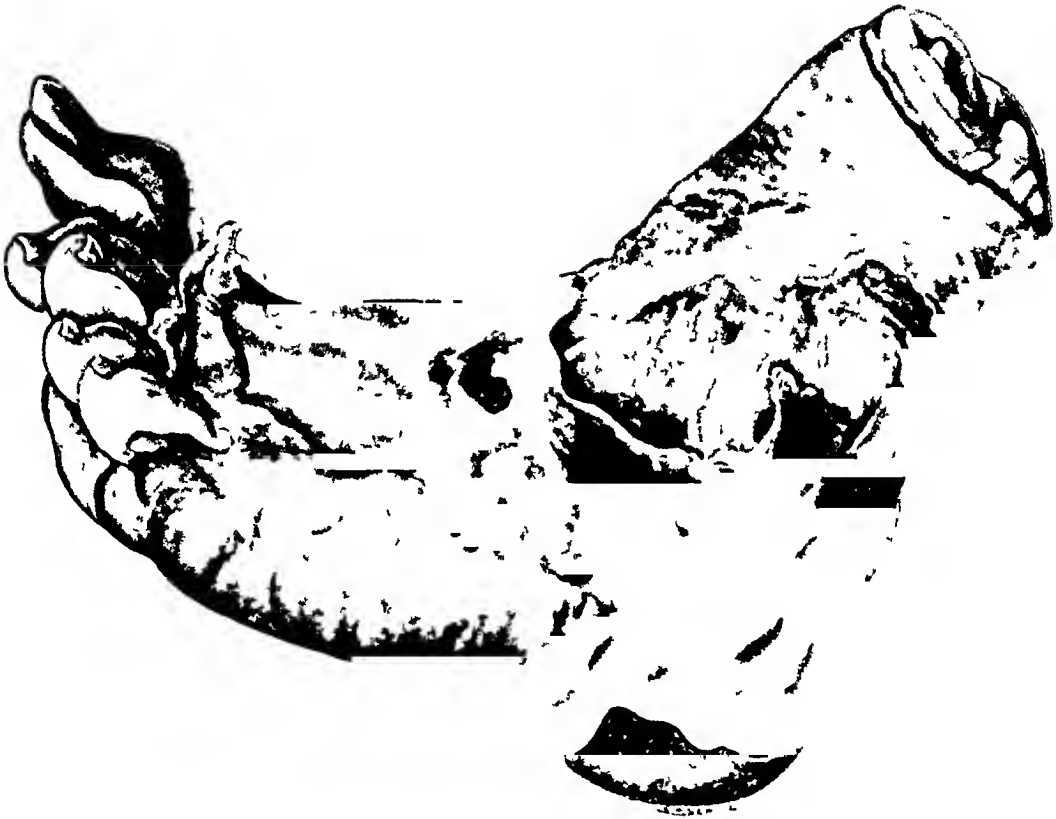
A left foot removed above the level of the line of demarcation

The foot is in a condition of moist gangrene and the skin is loose and discoloured. At the level of the ankle-joint there is a broad line of demarcation exposing the subcutaneous fat.

Museum of University College Hospital 612

CLINICAL HISTORY.—The patient was a man aged 39 who slept for a week in a wagon with wet boots on. His feet became painful, swollen, red and finally black. The urine was normal. Amputation two weeks later.

MOIST GANGRENE



TRENCH FOOT

A left foot drawn from the dorsal aspect

The foot has become gangrenous as the result of long-continued exposure to cold and wet. A line of demarcation has formed above the ankle.

Hunterian Museum R.C.S. Army Medical Collection, 1544 B

Clinical History—The patient was admitted to No. 9 General Hospital Rouen on April 3, 1916, with a history that he had been in the trenches for six days, that the weather had been cold with frost and snow, and that there was much water in the trenches.

The right foot was gangrenous as high as 2 in. above the ankle in front and to the heel behind. The gangrene was of the moist form and was very offensive. The leg was greatly swollen and very painful. The left foot was also gangrenous, but there was comparatively little pain on that side. The patient died five days later after a severe 'cardiac attack' with cyanosis and Cheyne-Stokes breathing. At the time of death the gangrene had extended to the knee on the right side.

Numerous blebs containing bloody fluid were scattered over the posterior surface of the body as high as the neck.

Autopsy—Lungs congested. Atheroma of mitral valve and commencement of aorta. 'Cloudy swelling' of liver and kidneys.



HUNTERIAN MUSEUM R.C.S.,
ARMY MEDICAL COLLECTION
1544 B

S.P. SENZU

CARBOLIC GANGRENE

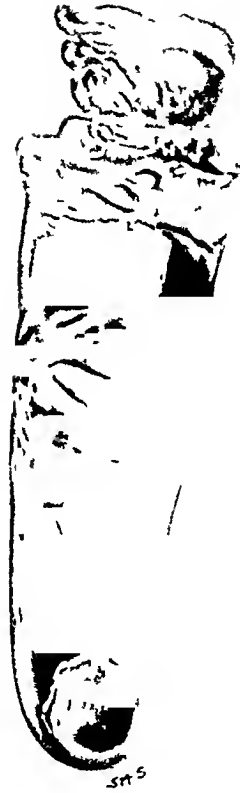
A finger drawn from the dorsal aspect

The whole surface of the skin and nail is black, but there is no evidence of ulceration or loss of tissue

Museum of St Bartholomew's Hospital, Z 45

CLINICAL HISTORY —The patient was a woman who had injured her finger. The injury was followed by suppuration. She wrapped the finger for one night only in lint which had been soaked in 1-20 carbolic lotion. The next morning the finger was livid and anæsthetic. After seven days a line of demarcation appeared and the finger was amputated.

CARBOLIC GANGRENE



MUSEUM OF ST BARTHOLOMEW'S HOSPITAL Z 45

LXXIII GAS GANGRENE

GAS gangrene was a common result of gunshot injuries in the war, especially those caused by high explosive shell under the conditions of trench warfare. In civil practice it is almost confined to the wounds caused by road accidents, especially when these have not been treated promptly by efficient surgical cleansing.

Gas gangrene is the result of infection of a lacerated wound of muscle by anaerobic bacteria, particularly *B. aerogenes capsulatus* (Nidekii). There is usually an associated infection by a hemolytic streptococcus.

The micro-organisms spread rapidly throughout the whole length of the injured muscles, but do not easily extend to the intact muscles alongside. The infected muscle is at first of an opaque brick-red colour and does not contract on mechanical stimulation. Later it turns brown and bubbles of gas appear in it and in the surrounding connective tissues.

If the bacterial invasion is well established by the time the case is first seen, the wound may be discharging a chocolate-coloured pus in which there are bubbles of gas. A brown discoloration of the skin spreads from the edge of the wound and may also extend over a considerable part of the limb even when the muscular invasion is at an early stage. This discoloration of the skin is an indication of a subcutaneous anaerobic infection, but is of no serious prognostic significance. The danger to the patient lies solely in the extent of the underlying muscular infection. In advanced cases, the limb becomes greatly swollen by oedema, cold, and tympanitic from the production of gas in large quantity. Shortly before death there is frequently an invasion of the blood-stream by hemolytic streptococci and gas-producing anaerobes, so that if there is a simple fracture in another limb, that limb may also become swollen and tympanitic from the production of gas.

GAS GANGRENE OF MUSCLE

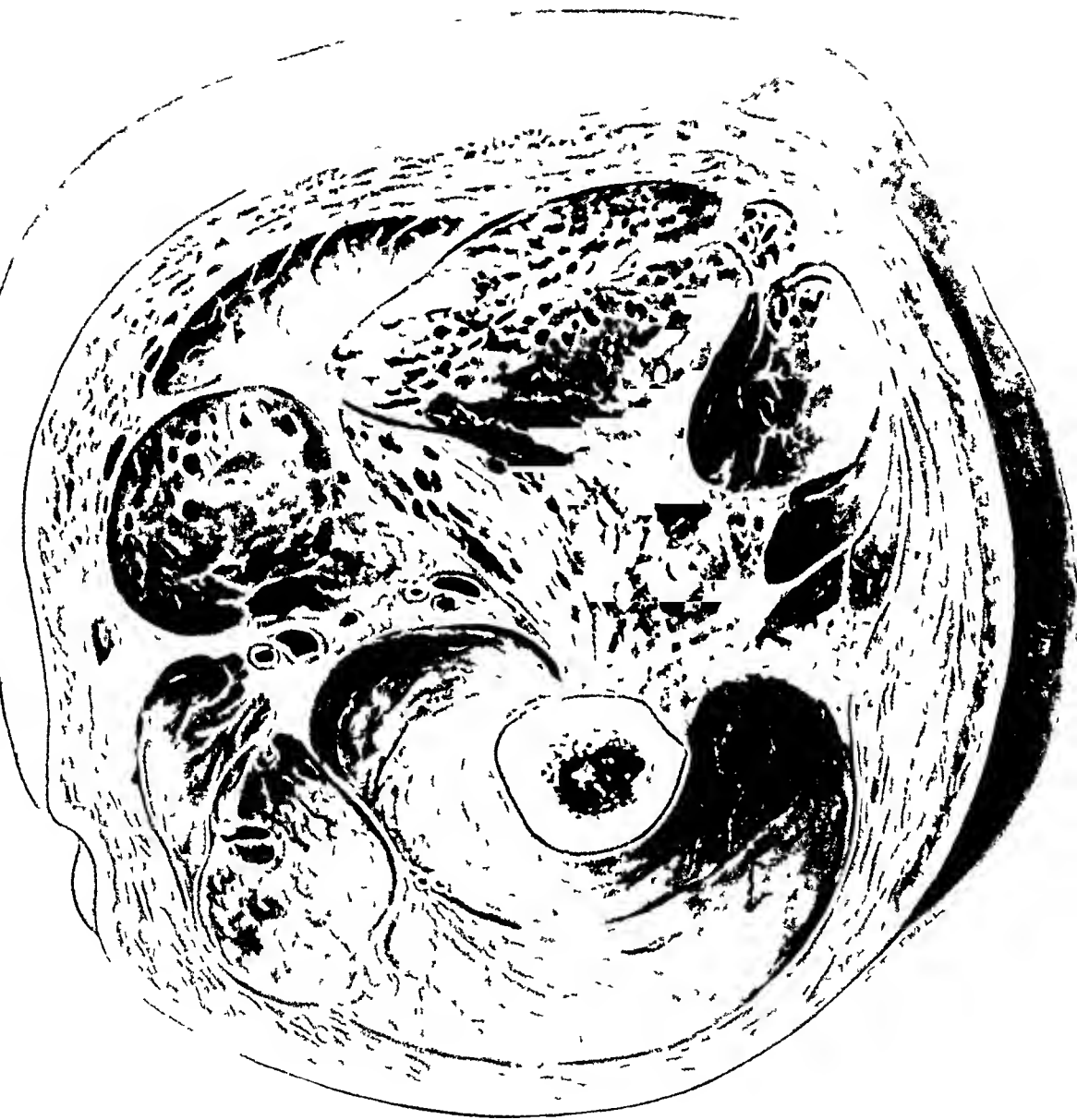
A transverse section of a right thigh

The muscles on the posterior aspect of the femur are emphysematous from gas gangrene and there are bubbles of gas in the neighbouring subcutaneous fat. The skin on the posterior surface of the limb is discoloured.

Hunterian Museum R.C.S. 2585.1

CLINICAL HISTORY —The patient was a soldier who was admitted to a General Hospital in Rouen in 1916 with a large foul gunshot wound about 9 by 6 in. on the back of the right thigh. Oedema and gaseous emphysema extended upwards to the abdominal wall. The length of time that had elapsed since the injury was unknown. The man died two and a half hours after admission.

GAS GANGRENE OF MUSCLE



HUNTERIAN MUSEUM R C S, 2588 1

LXXIV FIBROCYSTIC DISEASE OF BONE

ON February 6, 1934, a Joint Discussion on Fibrocystic Disease of Bone was held at the Royal College of Surgeons between the Section of Orthopaedics and the Section of Surgery of the Royal Society of Medicine.*

To illustrate the subject of this discussion a collection of specimens drawn from the Hunterian Museum and from the pathological museums of various hospitals was exhibited. It comprised seventy-one wet and dry specimens directly illustrating the subject of the discussion supplemented by a number of clinical photographs, X-ray pictures, and microscope slides and drawings. An additional thirty specimens illustrating miscellaneous cystic, fibrotic, and rarefying conditions of bone were presented.

The value of such a collection was so evident that it was decided to publish illustrations of the most characteristic specimens of each group in the *ATLAS OF PATHOLOGICAL ANATOMY*. The groups will be illustrated under the following headings:—

Solitary Cyst of Bone

Multiple Cysts of Bone

Osteoclastoma

Focal Fibrosis of Bone

Diffuse Fibrosis of Bone

Hyperparathyroid type of Fibrocystic Disease of Bone

* *Proc Roy Soc Med*, 1934, xxvii, No 7, pp 973-983

SOLITARY CYST OF FEMUR

The upper end of a femur divided by longitudinal section

The trochanteric region distal to the metaphysis is enlarged by a central cyst which is partially subdivided by septa. The cyst has a thin lining of dense fibrous tissue and contains red structureless material. There is a fracture of the neck of the femur.

Museum of University College Hospital 79A1

CLINICAL HISTORY —The patient was a boy aged 6 who had hurt his left groin by a fall one year before admission to hospital. Three months later he fell again and broke his left femur. The fracture united, but on the day before admission he fell and broke it again.

X-ray examination showed a globular enlargement of the neck and trochanter with a fracture running through it.

The specimen illustrated was removed by operation and the boy was treated on a double Thomas hip-splint for six months after which he was able to walk in a high boot without crutches.

(*P. M. Heath, Med Soc Trans*, 1911, ~~xxxv~~ 254)

SOLITARY CYST OF FEMUR



MUSEUM OF UNIVERSITY COLLEGE HOSPITAL 79A1

MULTILOCULAR CYST

A longitudinal section of a tibia with the surrounding soft parts

The whole of the shaft is expanded in fusiform shape by a fibrous tissue in which large cysts have developed. At the upper and lower extremities the edge of the fibrous part is sharply differentiated from the cancellous bone which remains. The cysts have a smooth lining and are crossed by the remains of blood-vessels. In the recent state they contained a yellow turbid fluid rich in cholesterol.

Hunterian Museum R.C.S. 7106

MICROSCOPIC STRUCTURE —The solid part below the larger cysts is composed of well-developed fibrous tissue. Sections of one of the septa between the cysts show multinucleated giant-cells and blood-pigment indicative of past extravasation. The cysts are lined by flattened connective-tissue cells.

CLINICAL HISTORY —The patient was a man aged 21 whose tibia had been broken just below the middle ten years before admission to hospital. The leg had ached more or less ever since and a lump remained near the site of fracture. For nine months before amputation his leg had increased in size but had not prevented him from working.

(*F. S. Eve, Trans. Pathol. Soc. Lond., 1888, xxxix 273*)

MULTILOCULAR CYST



HUNTERIAN MUSEUM R.C.S., 740 6

NO 38—SUPPLEMENT

OSTEOCLASTOMA OF FEMUR

The lower end of a femur divided by coronal section

The inner condyle of the femur is expanded by a deep-red growth which extends down to but not through the articular cartilage and is beginning to invade the opposite condyle. The growth is solid and stained by blood; its outline is clearly differentiated from the surrounding bone and there is no extension up the medullary cavity.

Museum of Westminster Hospital, 262B

MICROSCOPIC STRUCTURE —Osteoclastoma

CLINICAL HISTORY —The patient was a man aged 31 years who had complained of pain in the knee for eight months.

OSTEOCLASTOMA OF FEMUR



OSTEOCLASTOMA OF FEMUR

The upper half of a femur divided by longitudinal section

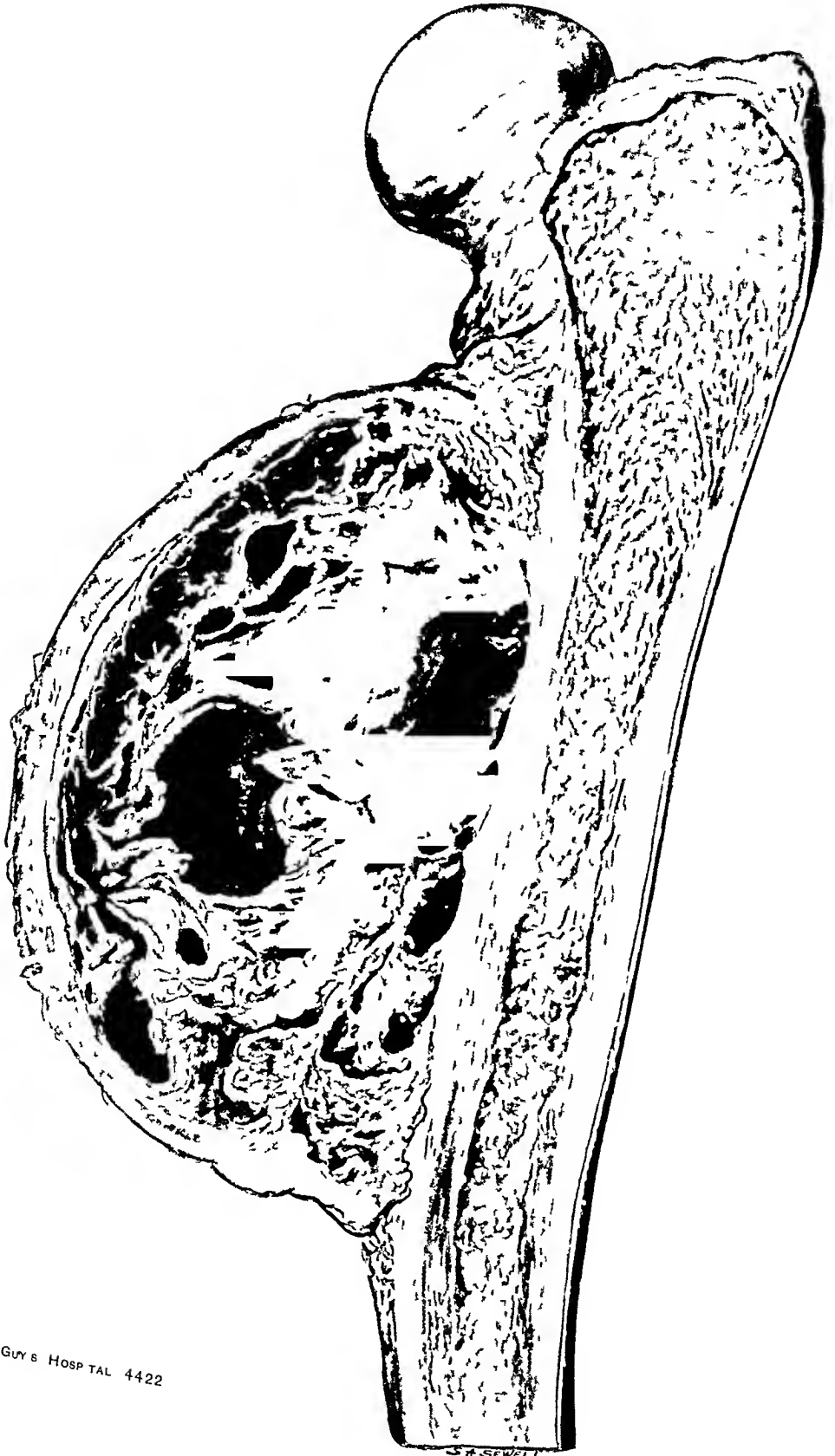
A cystic tumour is attached to the posterior aspect of the upper half of the femur. It has grown into the outer layer of the compact tissue of the shaft a thin layer of which covers it on its superficial aspect. It is composed of a number of thin-walled cysts of varying sizes separated by fibrous septa.

Museum of Guy's Hospital, 1422

MICROSCOPIC STRUCTURE.—Osteoclastoma

No clinical history

OSTEOCLASTOMA OF FEMUR



OSTEOCLASTOMA OF TIBIA

A sagittal section of the lower end of a tibia with the surrounding soft parts

For 3 in. above the epiphysal cartilage the tibia has been expanded by a myeloma and is composed of a thin shell of bone enclosing a central cavity. The interior is partly subdivided by incomplete septa but is otherwise smooth and is lined by a thin layer of growth red in colour from extravasated blood and sharply differentiated from the marrow of the shaft. At one point it has perforated the epiphysal cartilage. In the recent state the cavity contained a clear watery fluid tinged with blood.

The soft parts surrounding the expanded bone are displaced but not infiltrated.

Hunterian Museum, R C S, 1972 1

MICROSCOPIC STRUCTURE —The tumour consists of spindle-shaped cells among which are large numbers of multinucleated giant cells. It is highly vascular.

CLINICAL HISTORY —The patient was a girl aged 14, in whom an increasing swelling of the lower part of the left leg had been noticed for nine months. Pain was felt on walking.

(*H. A. Lediard, Proc Roy Soc Med (Clin Sect) 1911, 132*)

OSTEOCLASTOMA OF TIBIA



OSTEOCLASTOMA OF TIBIA

The upper end of a tibia divided by longitudinal section

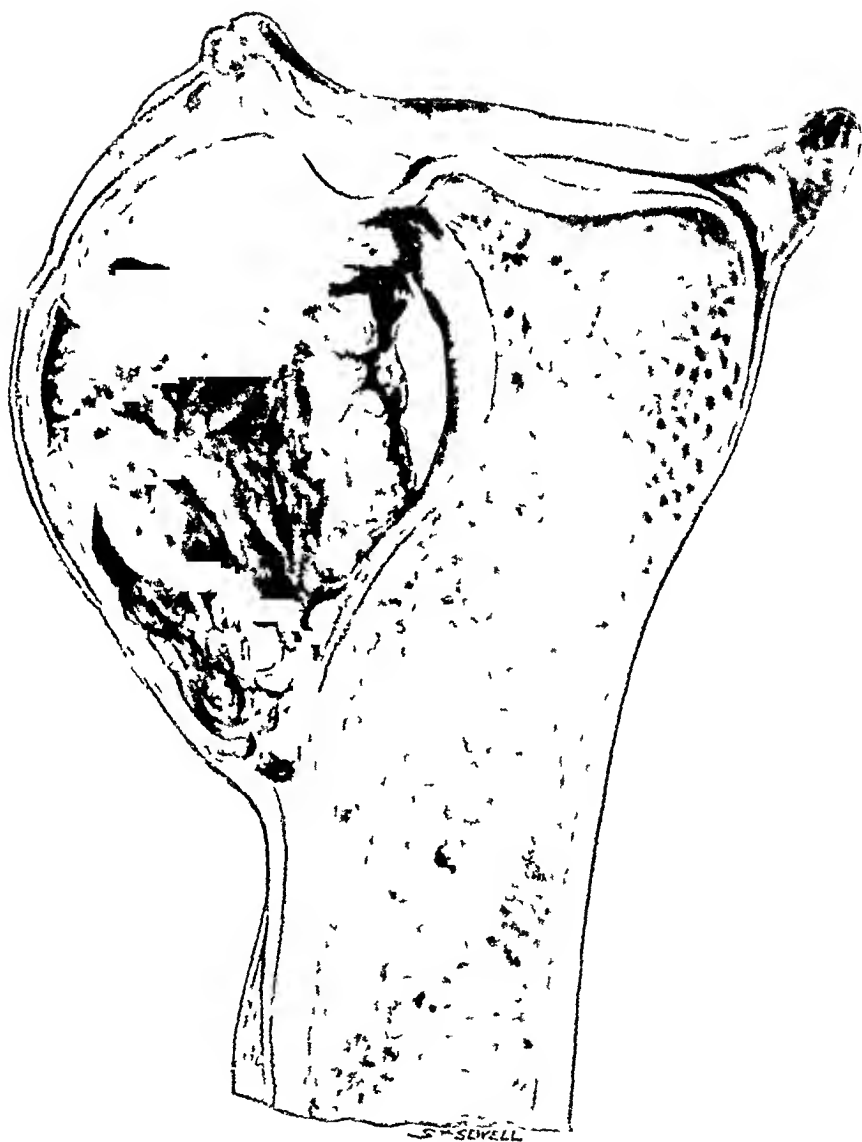
The posterior part of the head of the tibia is occupied by a single cyst which is lined by a dark-red ragged tissue and had contained sanguineous fluid. Posteriorly it projects into the popliteal space but has an incomplete shell of overlying new bone. The upper tibial epiphysis has fused with the shaft and the cyst extends to the articular cartilage. The appearance suggests central necrosis of an osteoclastoma.

Imperial Museum, R.C.S., 1971 1

MICROSCOPIC STRUCTURE —Osteoclastoma. The margin of the growth is bounded by a few bone lamellae undergoing osteoclasia on their deep aspect and osteogenesis on their superficial aspect. These in turn are bounded by a layer of fibrous tissue showing infiltration with small round cells.

CLINICAL HISTORY —The patient was a man, aged 19 years, who had complained of pain in the knee and later of swelling which limited flexion.

OSTEOCLASTOMA OF TIBIA



HUNTERIAN MUSEUM, R.C.S. 1971 1

OSTEOCLASTOMA OF FIRST METACARPAL

A thumb divided by longitudinal section

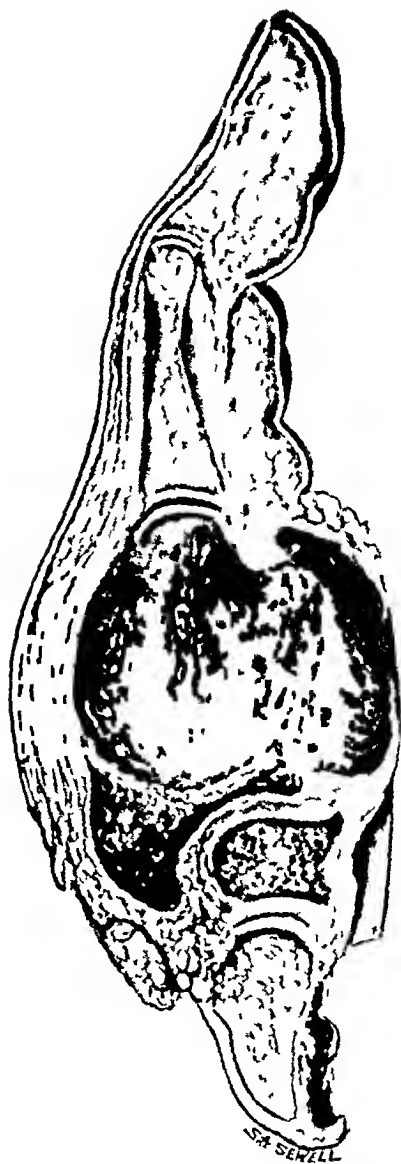
The first metacarpal has been greatly expanded by a growth which has destroyed practically the whole bone except over the distal articular cartilage. The tumour has extended beyond the original limits of the bone both on the palmar and on the dorsal aspects.

Museum of Westminster Hospital, 262A

MICROSCOPIC STRUCTURE —Osteoclastoma

No clinical history

OSTEOCLASTOMA OF FIRST METACARPAL



MUSEUM OF WESTMINSTER HOSPITAL, 262A

FOCAL FIBROSIS OF TIBIA

The lower two-thirds of a tibia and fibula with the interosseous membrane

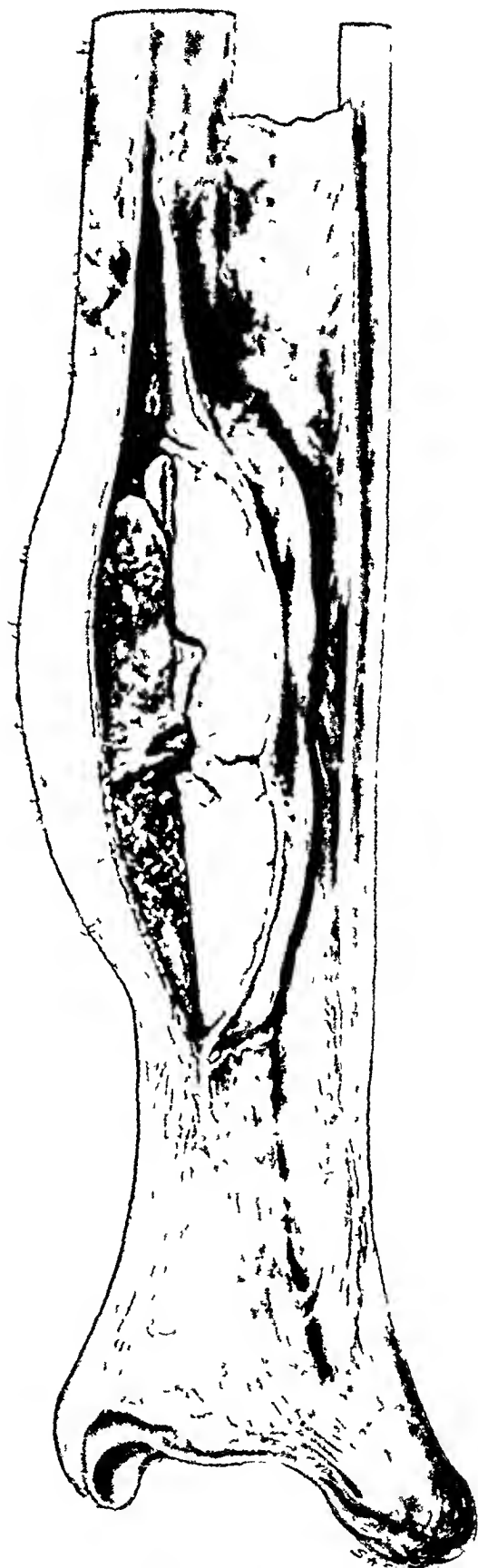
At the junction of its middle and lower third the tibia is expanded by a central, fusiform, encapsulated mass of firm fibrous tissue in which are scattered a few small spicules of bone

Museum of Guy's Hospital, 4419

MICROSCOPIC STRUCTURE —Well-formed fibrous tissue

CLINICAL HISTORY —The patient was a man aged 34 years. He had received a blow on the leg eighteen years before. This had caused a swelling which had increased in size during the eighteen months which preceded admission to hospital.

LOCAL FIBROSIS OF TIBIA



DIFFUSE FIBROSIS OF TIBIA AND FIBULA

The skeleton of a child's leg divided by longitudinal section

The middle third of both bones is bowed forwards and expanded by a mass of fibrous tissue of uniform appearance which entirely replaces both cortex and medulla and is sharply limited above and below

Museum of University College Hospital 79\2

Hunterian Museum R C S, 710 5

MICROSCOPIC STRUCTURE.—The mass consists of richly cellular fibrous tissue containing islands of bone which show osteogenetic activity in places and occasional lacunar absorption

CLINICAL HISTORY.—The patient was a girl aged 5 years. At the age of one year the right leg had been injured in a fall from a chair. This produced a fortnight's disability. A year and a half later increasing swelling of the leg was noticed

(*Bilton Pollard Trans Pathol Soc Lond 1885 XXXI, 388*)

DIFFUSE FIBROSIS OF TIBIA AND FIBULA



MUSEUM OF UNIVERSITY COLLEGE
HOSPITAL, 79A2
HUNTERIAN MUSEUM R C S , 740 5

HYPERPARATHYROID TYPE OF FIBROCYSTIC CHANGE

Two parathyroid tumours embedded in the thyroid gland and two renal calculi from the same case as the following illustration

London Hospital, 189/1930

Microscopic Structure—The left upper parathyroid body is surrounded by a capsule of dense collagenous fibrous tissue and elastic fibres which shows a few calcareous plaques in its inner aspect. Within this it is divided into small acini by a net of capillaries walled by a sheet of collagen. In three small areas the collagen is greatly swollen and hyaline whilst the capillary lumina are obliterated so that stout hyaline nets are formed enclosing small remnants of acini. Throughout much the greater part of the gland the acini contain polygonal cells with a deeply oxyphil eosinophil cytoplasm. This cytoplasm is almost always vacuolated but the vacuoles are usually very small, so that the cells typically have a solid granular appearance recalling hepatic cells. The cells measure in mean diameter 7 to 11.5 μ . Their nuclei are round or slightly oval. They usually measure 4 to 5 μ but variation in the size of the nuclei is conspicuous, many measure up to 7 μ and there are a very few of 9.5 μ . Most are very deeply stained. They show a stout membrane, a widely meshed net of chromatin, large rounded nodes and occasionally, one or more nucleoli. In sharp contrast to the main part of the gland are a few scattered groups of acini which contain smaller, greatly vacuolated cells. These cells measure from 6 to 9 μ . Their nuclei are usually 4 μ , rarely as large as 6 μ , the larger examples are less deeply stained. They show a basophil reticular cytoplasm and a basophil cell-membrane (pyronin-methylgreen). Occasionally a faintly stained oxyphil substance can be seen in the meshes of the reticulum. Most of the cells are very diopsical showing little or no cytoplasm except the cell-membrane. Some of these acini contain a few of the deeply oxyphil cells also.

In the adjacent thyroid gland most of the acini are large and filled with colloid, whilst the epithelial cells are sharply defined and flattened. In other acini there is little or no colloid, and the cells are ill-defined.

HYPERPARATHYROID TYPE OF FIBROCYSTIC CHANGE.



LONDON HOSPITAL, 489/1930

HYPERPARATHYROID TYPE OF FIBROCYSTIC CHANGE

A portion of the skull and the right humerus divided by longitudinal section from the same case as the preceding illustration

The right humerus is deformed and shortened the shaft is expanded by cystic areas over which the cortex has practically disappeared. The calvarium is greatly thickened and the cut surface finely porous.

London Hospital, 192/1930

MICROSCOPIC STRUCTURE —The cortical bone of the shaft of the humerus is thin and is largely replaced by a layer of fibrous marrow in which are trabeculae with numerous osteoclasts in Howship's lacunae with osteoid zones covered by a layer of closely packed large osteoclasts in the fibrous marrow. The capillaries are slightly enlarged and there are a few hemorrhages and many groups of cells loaded with iron pigment. The medulla contains fat.

CLINICAL HISTORY —The patient was a married woman aged 49 years. Eight years before admission to hospital her back began to bend and her shoulders became rounded so that her total height gradually diminished. For four years she had had multiple spontaneous fractures which gave rise to deformities of the limbs. Ultimately she became bedridden. She was of normal intelligence but totally disabled, greatly deformed, breathless and wasted. There was an angular curve in the spine at the level of the 5th thoracic vertebra with gross deformity of both upper limbs and of the thorax. The legs were crossed owing to mal-union of a fracture of the left femur.

X-RAY —Poverty of shadows of all bones examined, loss of detail and stippling. Atrophic changes, pale cyst-like spaces, and old fractures in bones of limbs. Kyphosis, collapse of ribs, and tri-radiate pelvis. Calvaria greatly thickened, blurred, and coarsely mottled. Large calculi in right kidney.

PROGRESS —Progressive emaciation and exhaustion. Serum calcium 13.3 mgrm per 100 c.c. Died following further spontaneous fractures.

AUTOPSY —Bronchopneumonia. Gross changes in bones and parathyroid glands. Three large irregular calculi in right kidney with hydronephrosis and dilated ureter. No calculi in left kidney. Heart, liver, and spleen wasted. Larynx, trachea, oesophagus, suprarenals, pancreas, uterus, ovaries, and pituitary normal. No macroscopical evidence of metastatic calcification found in any organ.

COMPOSITION OF RENAL CALCULI —The calculi consist principally of calcium phosphate.

HYPERPARATHYROID TYPE OF FIBROCYSTIC CHANGE



LONDON HOSPITAL 482/1930

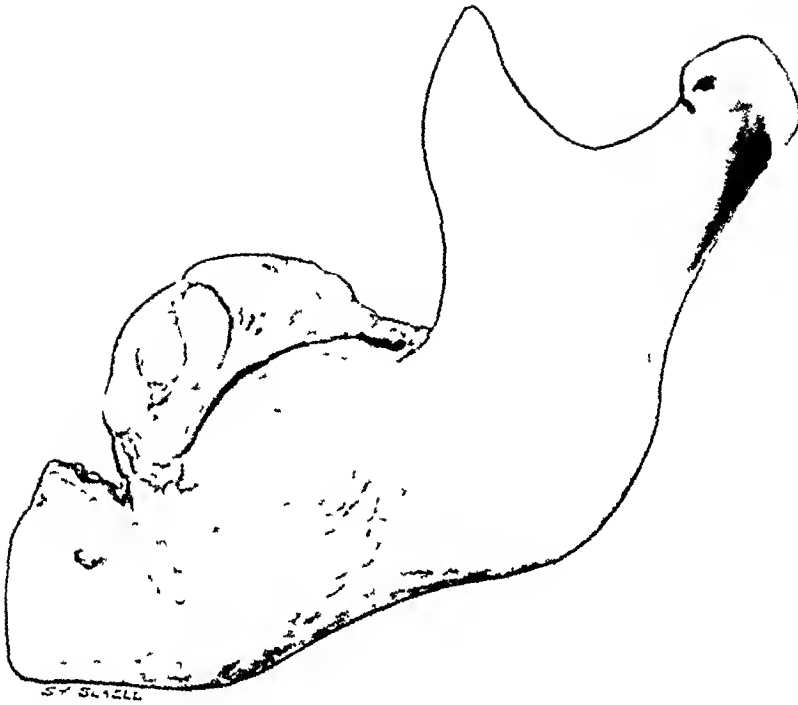
HYPERPARATHYROID TYPE OF FIBROCYSTIC CHANGE



Longitudinal section of entire shaft near centre of humerus, to show the strict limitation of the fibrosis to the altered corticals. Generalized osteitis fibrosa. Muller's fluid followed by nitric acid, Weigert's non-haematoxylin and van Gieson.

London Hospital, 482, 1930

OSTEOCLASTOMA OF MANDIBLE



The left ramus of a lower jaw with an adjoining part of the body 5 cm in length

Immediately in front of the ramus the body of the jaw with the exception of its lower border is expanded by the growth of a myeloma which has protruded above through the alveolar process so as to form a second superficially lobulated, and somewhat flattened mass covered with mucous membrane and overhanging that part of the growth which is enclosed by the bone

Museum of University College Hospital, S A 1

MICROSCOPIC STRUCTURE —Osteoclastoma

CLINICAL HISTORY —The parts were removed by Mr Liston

OSTEOCLASTOMA OF FEMUR

The lower part of a femur together with some of the surrounding muscles divided by longitudinal section

Immediately above the epiphyseal line the femur is expanded by a central tumour which is yellowish-red in colour necrotic in the centre and cystic in parts. It has extended for a short distance up the medullary cavity and into the popliteal space posteriorly, but has not penetrated the epiphyseal cartilage

Museum of Guy's Hospital, 4428

MICROSCOPIC STRUCTURE --Osteoclastoma with formation of cysts by myxomatous degeneration

CLINICAL HISTORY --The patient was a boy aged 15 years, who had had a painless swelling increasing in size for five months

The specimen was removed by amputation

OSTEOCLASTOMA OF FEMUR



MUSEUM OF GUY'S HOSPITAL 4428

S. J. SEWELL

OSTEOCLASTOMA OF TIBIA

The lower end of a tibia divided by coronal section

The inner part of the lower extremity is expanded by a central tumour which extends into the malleolus and reaches to, but does not penetrate the articular cartilage. Above and on the outer side its outline is sharply differentiated from the surrounding cancellous tissue. The tumour does not extend through the periosteum nor up the medullary cavity. Its cut surface shows a large number of small cysts which contained a blood-stained fluid.

Museum of University College Hospital, 85 A 1

MICROSCOPIC STRUCTURE — Osteoclastoma

No clinical history

OSTEOCLASTOMA OF TIBIA



MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 85 A 4

FIBROCYSTIC DISEASE OF FEMUR

The lower end of a right femur divided by longitudinal section

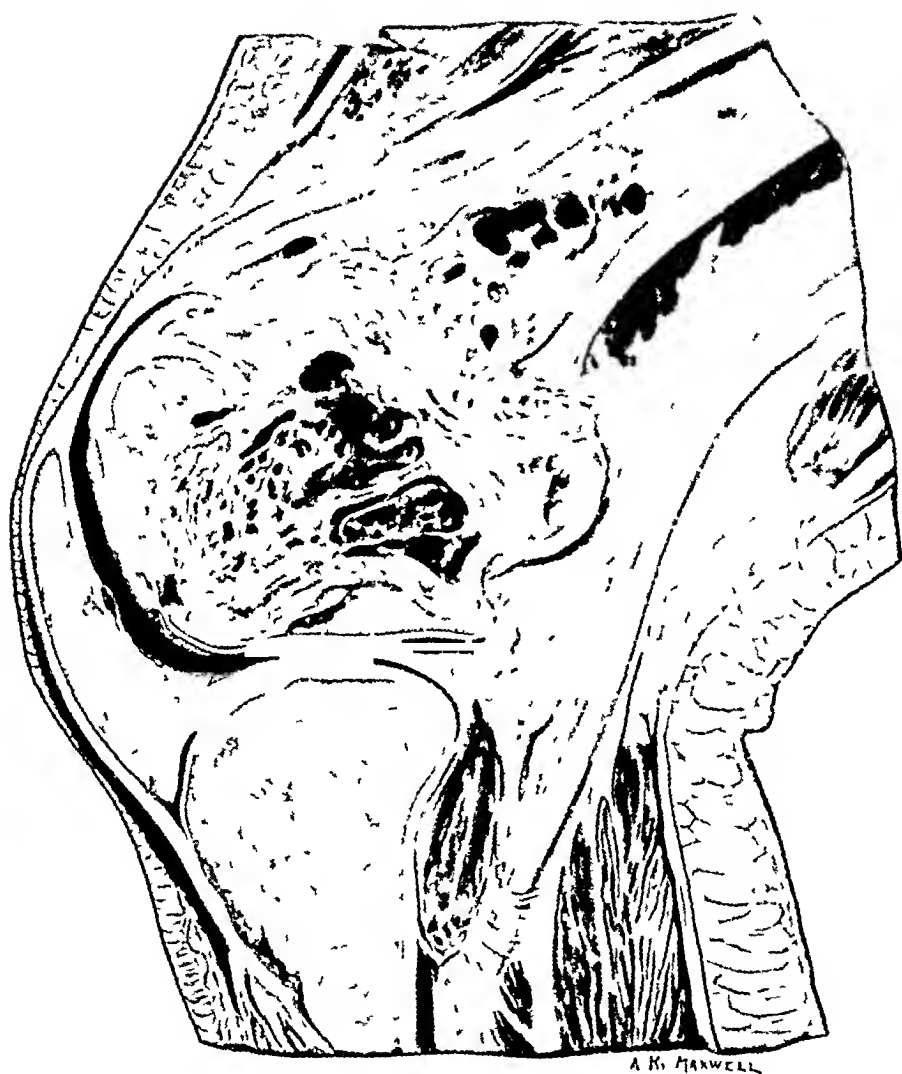
The lower extremity of the femur is expanded by fibrous tissue honey-combed with cystic spaces of varying sizes, some of which contain blood-clot. The articular cartilage is intact but its surface is somewhat deformed. The cystic formation extends upwards for two inches into the medullary canal replacing the marrow and posteriorly forms a rounded projection in the popliteal space.

Hunterian Museum R.C.S., 7101

MICROSCOPIC EXAMINATION—The septa separating the cysts are composed of firm fibrous tissue which is abundantly cellular. Towards the centres of the cysts the fibres have undergone a degenerative change resulting in their division into fibrils separated by a liquid substance and staining with haematoxylin. The cysts appear to have arisen from the complete liquefaction of these fibrils. Bone is present but no leucocytic exudate.

CLINICAL HISTORY—The patient was a girl, aged 17 years, who in November, 1920, fractured her right femur. The fracture united after treatment. Eight months later her right knee gave way under her. On admission to hospital a large swelling was found at the back of the knee, this was scraped out and was considered to be a myxoma. Twelve months later there was painful swelling of the knee which was treated by scraping and X rays. The tumour was reported to be a fibrosarcoma. After a second relapse the patient was re-admitted in December, 1923, with a swelling of the lower end of the femur and $2\frac{1}{2}$ in. of shortening. The leg was amputated and the patient made a good recovery.

FIBROCYSTIC DISEASE OF FEMUR



HUNTERIAN MUSEUM, RCS 740 1

SOLITARY CYST OF HUMERUS

The upper end of a humerus divided by longitudinal section

The cyst is metaphyseal in position and is partially subdivided by septa. It is lined by a thin layer of fibrous tissue and in the fresh state contained a clear dark straw-coloured fluid.

Hunterian Museum R.C.S., 1970 1

MICROSCOPIC STRUCTURE —The lining consists of fibroblastic tissue containing patches of osteoid tissue, in places osteoclasts are in contact with this. The histological appearance closely resembles that of osteogenic sarcoma except that the cells tend to show greater uniformity and that multinucleate cells of malignant type and mitosis are not apparent. The septa are composed of fibrous tissue containing an occasional osteoclast.

CLINICAL HISTORY —The patient was a boy, aged 7 years, who fell whilst running on level ground and complained of having hurt his left shoulder. Examination at that time showed nothing abnormal. During the following seven months he was never able to use the arm quite freely and complained that it hurt him if it were suddenly jerked. At the end of this time he had a second fall and complained that he had hurt the arm again in the same place.

On examination the upper end of the left humerus was enlarged and was tender on firm pressure. There was no redness or oedema of the skin over the swelling. A skiagram confirmed the diagnosis of tumour, and the limb was removed by amputation through the neck of the scapula. Recovery was uneventful.

SOLITARY CYST OF HUMERUS



HUNTERIAN MUSEUM R C S , 1970 1

MULTILOCULAR CYST OF HUMERUS

One-half of the upper end of a humerus divided by longitudinal section

The metaphysis and upper end of the shaft are expanded by a multilocular cyst which is bounded by the epiphyseal line above and is sharply differentiated from the medullary canal below. There is a recent fracture through the lower part of the cyst.

The cyst is lined by fibrous tissue and subdivided by a number of fibrous partitions. In the recent state the cavity contained a pink fluid.

Hunterian Museum, R C 5 710 2

MICROSCOPIC STRUCTURE —The lining and septa of the cyst are composed of spindle-celled fibrous tissue containing multinucleate giant cells.

CLINICAL HISTORY —The patient was a boy aged 19 who hurt his shoulder whilst leaving a motor-bus in motion. A skiagraph furnished evidence that the humerus was fractured at the surgical neck, but the bone at the seat of the injury exhibited a shadow which suggested the presence of an endosteal sarcoma. The upper third of the humerus was excised.

MULTILOCULAR CYST OF HUMERUS



HUNTERIAN MUSEUM, R C S 7402

FIBROCYSTIC DISEASE OF HUMERUS

Part of the middle of the shaft of a humerus

For nearly its whole length the wall of the bone is thinned and on one side is bulged out by the formation of a well-defined cavity thinly lined with connective tissue from which delicate translucent lamellae pass across the space. The effusion of blood is the result of operative manipulation. At the back of the specimen there is shown an incomplete fracture of the attenuated bone, in connection with which an irregular formation of callus has taken place. The whole of the osseous tissue is of normal hardness. At the lower end of the section immediately below the cyst there is an irregular space, which in the other half of the specimen is occupied by a mass of fibrous tissue 1 cm in its longest diameter.

Humertum Museum, R.C.S. 1806.1

MICROSCOPIC EXAMINATION—The lamellae consist of finely fibrillar connective tissue. There are no multinucleated giant cells, nor is there any indication that the fibrous tissue has resulted from the decalcification and metaplasia of osseous trabeculae.

CLINICAL HISTORY—The patient was a schoolboy aged 12, who had twice suffered fracture of the middle of the shaft of the right humerus, in each case the result of throwing a cricket ball. The X-ray showed a localized cyst in the middle of the shaft with recent fracture. A portion of the shaft 3 in long was resected subperiosteally and the continuity of the bone restored by inserting a massive beef bone-graft into each fragment. The two grafts being bolted together in the gap. Three months later he was able to play games, and within eight years he had grown to be 6 ft 1 in in height and won the contest for putting the weight at the inter-varsity sports, using for this purpose the arm which had been mended.

FIBROCYSTIC DISEASE OF HUMERUS



HUNTERIAN MUSEUM R C S 3806 1

DIFFUSE FIBROSIS OF TIBIA AND FIBULA

A tibia and fibula, parts of which have been removed to show the structure of the bone

Both bones show bowing inwards and forwards, the fibula more so than the tibia. The upper two-thirds of each diaphysis is thickened and congested, and shows on section replacement of the whole bone structure by a firm uniform tissue which obliterates the medullary canal, in the upper part no portion of the corticæis persists but lower down it remains as a thin shell

Hunterian Museum R.C.S. 7405

MICROSCOPIC STRUCTURE—Highly vascular fibrous tissue, containing islands of osseous tissue. In places osteogenesis is occurring. Osteoclasts are inconspicuous.

CLINICAL HISTORY—The patient was a man aged 20 who complained of weakness of the left leg and inability to walk without support since the age of 2 years. The femur had been affected during life by bowing and had shown the same radiological abnormalities as the tibia and fibula. The femur had been fractured twice and the tibia once.

DIFFUSE FIBROSIS OF TIBIA AND FIBULA



HUNTERIAN MUSEUM, R C S, 740 8

HYPERPARATHYROID TYPE OF FIBROCYSTIC CHANGE

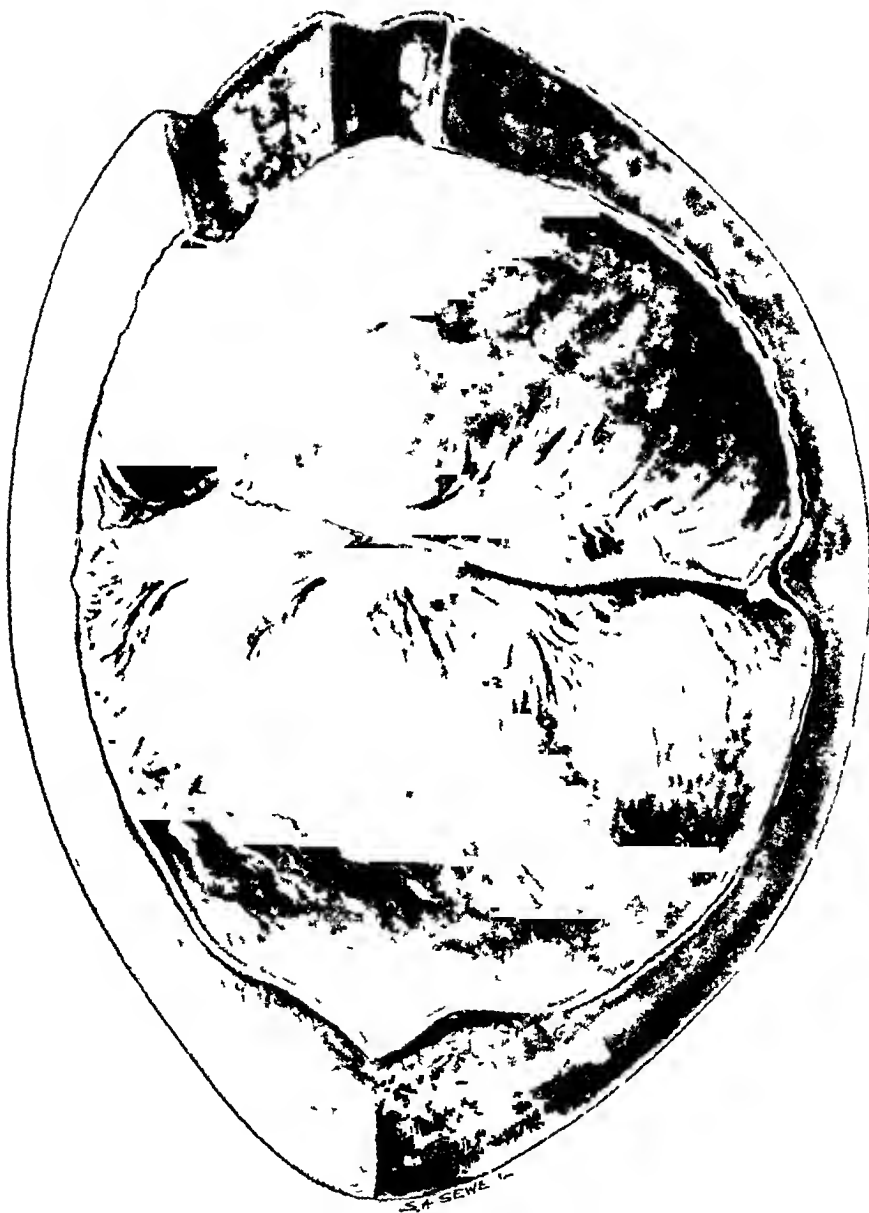
The anterior part of a calvaria

The bone is enlarged in all parts to a thickness of $\frac{1}{2}$ to $\frac{3}{4}$ in. Its structure is uniform. The distinction between the diploe and the external and internal table is absent and the normal osseous substance has been replaced by an extremely close cancellous tissue almost amounting to actual compact bone in places. Except for the denser areas the surface of the bone as seen through the unaltered pericranium and dura is congested.

Hunterian Museum R.C.S. 56081

MICROSCOPIC EXAMINATION — Dense cancellous structure in which the marrow is entirely replaced by a very cellular fibrous tissue. Numerous osteoclasts are present in places and a single layer of osteoblasts covers many of the trabeculae.

(Strangeways Collection 1927)



HUNTERIAN MUSEUM, RCS, 3808 1

NO 39—SUPPLMENT

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HYPERPARATHYROID TYPE OF FIBROCYSTIC CHANGE

The upper part of a left femur from the same case as No. 35081 divided longitudinally

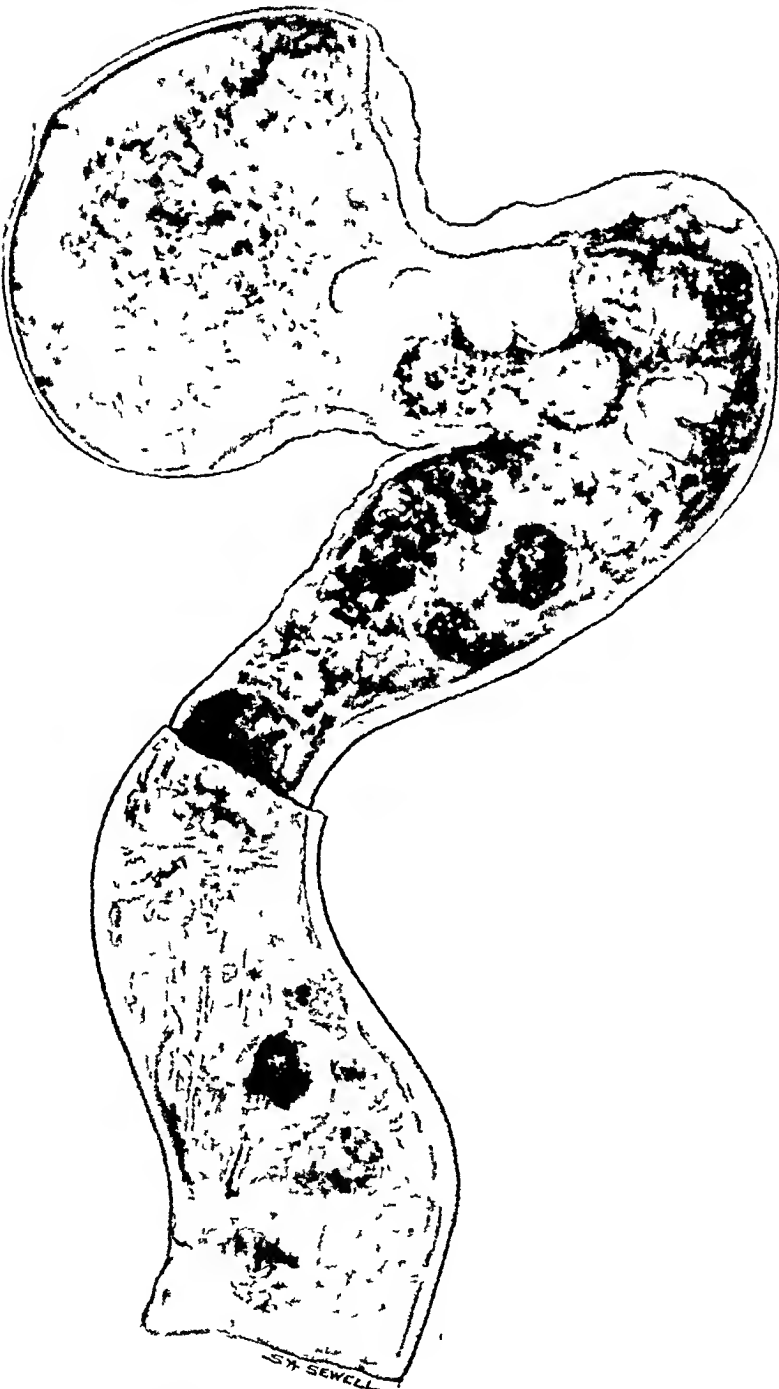
The head of the femur is flattened and depressed below the normal level the neck is short and atrophied and the upper part of the shaft is bent into a sigmoid form. The cortical layer of the shaft is very thin and the medullary cavity is correspondingly enlarged. The marrow is of the red variety and has scattered through it small islands of fibrous tissue in some of which are spicules of bone. The osseous tissue of the head and neck exhibits the same close cancellous structure as in the calvaria and in one-half of the section several nodules of hyaline cartilage are present in the neck and great trochanter.

Hunterian Museum R.C.S. 35081

MICROSCOPIC EXAMINATION - There is considerable rarefaction of the osseous tissue with development of numerous tracts of richly cellular and vascularized fibrous tissue in which are minute spicules of newly-formed bone the marrow in the affected areas being entirely replaced. Immediately beneath the articular cartilage is an almost continuous but very narrow, layer of tissue with the structure of fibrocystic disease.

(Strangeways Collection 1927)

HYPERPARATHYROID TYPE OF FIBROCYSTIC CHANGE



HUNTERIAN MUSEUM R C S 3808 3

HYPERTHYROID TYPE OF FIBROCYSTIC CHANGE

A right tibia and fibula from the same case as No. 35081

The shafts of the bones are more slender than normal and have a necked convexity towards the section made in the middle of the shaft of the tibia shows the marrow to be of a deep red color and the surrounding osseous tissue to have a close cancellous instead of its usual compact structure. There is a united fracture in the upper third of the shaft of the tibia.

Harvard Museum, No. 35081

Microscopic Examination. In the irregular new bone formation around the medullary cavity the Haversian canals and arteriovenous spaces are filled with a richly cellular and well vascularized fibrous tissue.

Clinical History. The patient was a girl, aged 19 years, whose family history did not include any disease of bones. She was admitted to hospital in May, 1909, complaining of swelling of the knees for the past four years. She had been treated at St. Thomas's Hospital for eight months in 1907. Osteotomy of one femur had been performed in October, 1906.

On admission there was extreme deformity of all the long bones and of the pelvis and scapula. The limbs were short and the movements of the right joints were limited. In September, 1910, a spontaneous fracture of the right tibia occurred. It was treated with splints, but there was no firm union by February, 1911.

She was examined again in October, 1911, and found to have bending of all the long bones and lateral curvature of the dorsal spine. The phalanges of the hands and feet were short, but the joints moved freely. The phalanges of the hands and feet were short, but the joints moved freely. The phalanges of the hands and feet were short, but the joints moved freely.

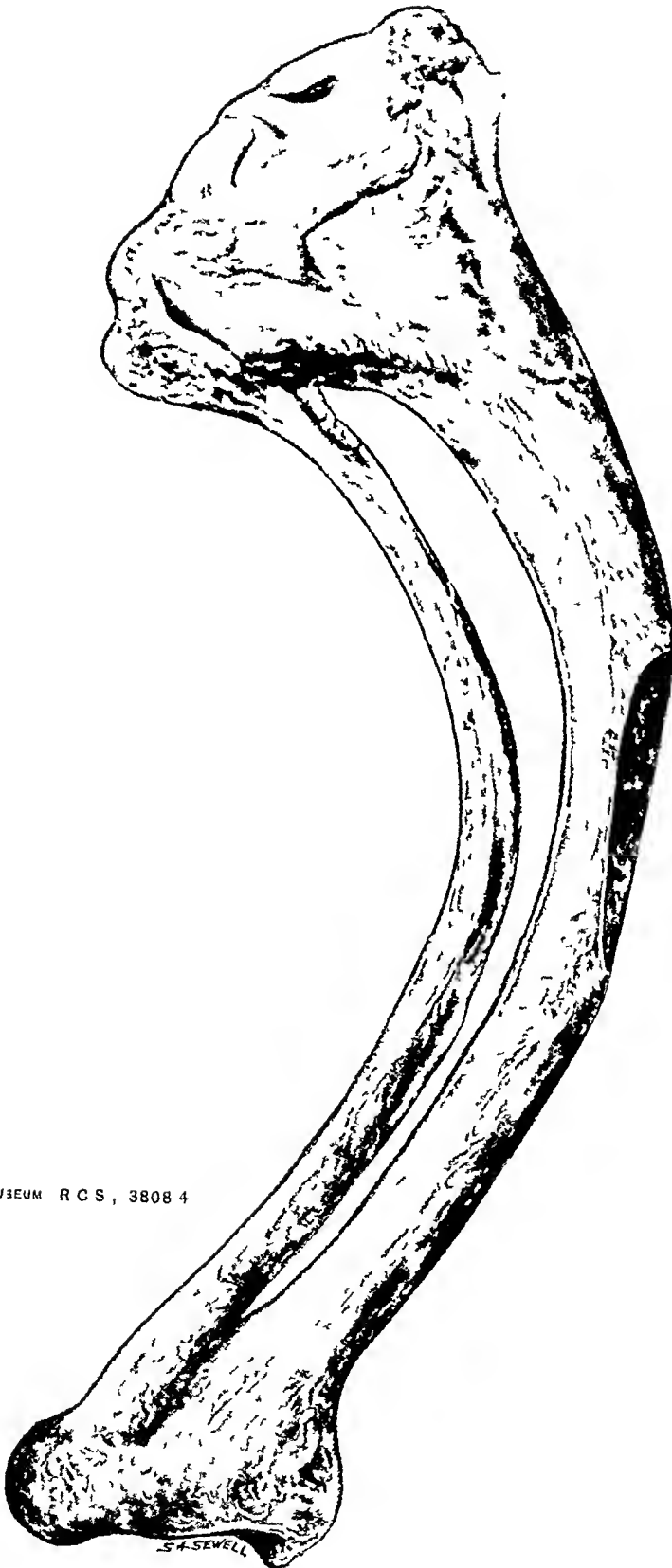
In August, 1912, she fell and complained of pain and tenderness at the site of the old fracture of the tibia. No abnormal mobility or crepitus was found on examination.

She died in October, 1912, after an attack of perforative appendicitis.

Atopsy.—The body was 3 ft. 9 in. high. Apart from the skeletal changes there is no record of disease. The pituitary, thyroid, suprarenal, pancreas, and thymus were normal.

(Strange's Collection, 1927)

HYPERPARATHYROID TYPE OF FIBROCYSTIC CHANGE



HUNTERIAN MUSEUM R C S , 3808 4

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- 1 *Hunterian Museum, R.C.S.* 19701—Upper end of humerus. Illustrated in *Atlas* No. 39, p. 425.
- 2 *Kings College Hospital*, A742—Upper end of humerus. Microscopic examination shows fibrocystic disease. No slide available. Boy, aged 11. Recent fracture.
- 3 *Hunterian Museum, R.C.S.* 7103—Upper end of humerus. The wall consists of an outer compact layer and an inner layer of cancellous tissue containing patches of fibrous tissue. The cavity is filled with a red, solid material. Three successive fractures.
- 4 *Kings College Hospital*, A743—Upper end of humerus. A resected portion of the upper third of the humerus showing slight expansion by an irregular cystic cavity which lay distal to the metaphysis showed trabeculation in the sklerium and had resulted in fracture. Microscopic examination shows osteoclastomatous tissue with very active bony absorption of the marginal bone. The osteoclastomatous tissue is intersected by fibrous bands containing osteoid tissue outlined in places by rows of osteoblasts. Girl, aged 16. The upper portion of the diseased area was not removed, and was observed seven years later to have extended and to be situated in a relatively more distal position.
- 5 *Royal Free Hospital*, 96/571—Upper quarter of humerus. The cyst is lined by fibrous tissue and contains gelatinous material. Boy, aged 3. Ten weeks' pain in the right shoulder following a fall on the hand. Four weeks' further pain and some loss of function after a fall on the arm.
- 6 *Hunterian Museum, R.C.S.* 38061—Middle of shaft of humerus. Illustrated in *Atlas* No. 39, p. 429.
- 7 *Charing Cross Hospital*, 1332—Upper end of radius. The cyst is metaphyseal in position and had contained a yellow fluid. Microscopic examination shows that the lining consists of fibroblastic tissue containing relatively large islands of osteoid tissue, osteogenesis is apparent in parts and a few osteoclasts are in contact with the osteoid tissue. Patient, aged 22.
- 8 *University College Hospital*, 79A1 (11262)—Upper end of femur. Illustrated in *Atlas* No. 38, p. 395.
- 9 *St Bartholomew's Hospital*, A679—Lower end of fibula. The cyst is metaphyseal in position and possesses a scanty lining composed of cellular tissue containing multinucleated giant cells. The lining is continuous with several thin septa. The cyst had contained a blood-stained fluid. Girl, aged 16. Twenty-two months' history of increasing swelling with pain on walking.
- 10 *London Hospital*, 180/1932—Upper end of fibula. The cyst is metaphyseal in position and possesses a scanty fibrous-tissue lining containing a few osteoclasts. It had contained an orange-coloured fluid. Boy, aged 20. Pain.
- 11 *St Bartholomew's Hospital*, A674—Outer third of clavicle. The cavity, which reaches the end of the bone, is intersected by fibrous septa and contains zones consisting of osteoclastomatous tissue. Girl, aged 6. Six weeks' history of increasing redness, tenderness, and swelling, no pain.

* At a recent meeting (Feb. 1934) of the Orthopaedic and Surgical Sections of the Royal Society of Medicine, a number of specimens illustrating cystic conditions of bone were exhibited. For the sake of future workers on this subject, all these specimens are here catalogued and a number drawn.

FIBROCYSTIC DISEASE OF BONE CATALOGUE

- 12 *Westminster Hospital* 249A—Mandible. The right half of the mandible the body of which is occupied and expanded by a cyst extending from the symphysis to the angle. Section of the wall shows fibrous tissue in the superficial part of which are bone lamellæ; the deep aspect of these shows very active lacunar absorption, while the superficial aspect shows the formation of osteoid tissue by apposition. No epithelial elements or inflammatory changes are apparent. (Child. (No history).)
- 13 *St. Mary's Hospital* 98 520*—Upper end of tibia split. Immediately distal to the epiphyseal line it is expanded by an irregular cystic cavity which is subdivided by many coarse trabeculae stretching between masses of firm solid tissue. Microscopical examination shows that the contents of the bony cavity consist partly of osteoclastomatous tissue and partly of fibrous tissue which is laying down scanty osteoid tissue in places. Both zones show cystic spaces containing blood. The marginal trabeculae show osteoclastic absorption on their deep aspect and osteogenesis on their superficial aspect. Girl aged 11. Constant pain had been present since an injury thirteen months before.
- 14 *St. Mary's Hospital* 98 520†—Lower end of femur the outer condyle bone expanded by a rounded cyst about 5 cm. in diameter containing a mucobronous lining giving off many film like septa. Microscopical examination of the subchondral zone shows fibrous tissue partly separated from the articular cartilage by normal bone lamellæ on the deep surface of which osteogenesis is occurring within the fibrous tissue. Adult.

MULTIPLE CYSTS IN BONE

- 15 *Hunterian Museum, R.C.S.* 740 2—Upper end of humerus. Illustrated in *Atlas* No. 19, p. 127.
- 16 *Guy's Hospital* 443B₂—Upper end of shaft of humerus. The cysts contain blood and the tissue in which they lie consists partly of fibrous tissue containing bone lamellæ densely outlined by osteoblasts and a very few osteoclasts and partly of fibroblastic tissue devoid of lamellæ and containing a few osteoclasts. Boy, aged 4. Swelling which increased in spite of curettage. Recovery followed subperiosteal resection.
- 17 *Guy's Hospital* 443B₂—Scapula. The lining of the cysts is composed of cellular fibrous tissue containing spicules of bone and the bony walls show marginal osteoclastic activity. Boy, aged 9. Increasing swelling first noticed one week after a fall sustained four years before operation.
- 18 *Westminster Hospital* 262B—Lower end of femur. Illustrated in *Atlas* No. 35, p. 399.
- 19 *Hunterian Museum, R.C.S.* 740 6—Longitudinal section of tibia. Illustrated in *Atlas* No. 38, p. 397.

OSTEOCLASTOMA

- 20 *University College Hospital* 85A3 (12215)—Lower end of femur. Microscopical examination shows the structure of an osteoclastoma. Man, aged 39. Pain for seven months and swelling for four months.
- 21 *London Hospital*, 529/1927—Lower end of femur. Woman, aged 39.
- 22 *St. Thomas's Hospital* 661A—246B—Lower end of femur (macrated). Microscopical examination showed that the tumour responsible for the bone change was an osteoclastoma. Man, aged 26. Swelling had followed one month after an injury sustained five months previous to examination.
- 23 *Royal Free Hospital*, 98 711†—Upper end of tibia. Microscopical examination shows the structure of an osteoclastoma containing areas of necrosis and haemorrhage. Man, aged 28. Nine months' stiffness of the knee, two months' pain and swelling.
- 24 *Westminster Hospital* 262A—First metacarpal. Illustrated in *Atlas* No. 38, p. 407.

FIBROCYSTIC DISEASE OF BONE CATALOGUE

- 25 *Royal Free Hospital* 96744 — Scapula. Microscopical examination shows the structure of an osteoclastoma. Six weeks' history of pain and swelling over the left scapula.
- 26 *Royal Free Hospital* 92744¹ — Mandible. Microscopical examination shows the structure of an osteoclastoma. Boy, aged 9. Acute, pain and swelling noticed for one week.
- 27 *University College Hospital* 8411 (12680) — Mandible. Illustrated in *Atlas* No. 39 p. 117.
- 28 *Cancer Hospital* 1495 — Maxilla. Microscopical examination showed the structure of an osteoclastoma. Man, aged 42. Six months' increasing swelling of the left cheek. (Note: At post mortem examination renal calculi and what was taken for an accessory thyroid gland were found. The case is possibly therefore an example of hyperparathyroidism.)
- 29 *University College Hospital* 8411 (14714) — Maxilla. The bone is occupied by a partly cystic growth which protrudes into the antrum. The cysts contain blood clot. Microscopical examination shows the structure of an osteoclastoma. Boy, aged 6. Eight weeks' history of painless swelling.
- 30 *St. Thomas's Hospital*, 1629—2466 — Lower end of femur. The tumour contains multiple cysts and microscopic examination shows the structure of an osteoclastoma. Girl, aged 20. Three and a half years' pain—radiation treatment.
- 31 *Guy's Hospital* 4428 — Lower end of femur. Illustrated in *Atlas* No. 39 p. 119.
- 32 *University College Hospital* 8531 (12524) — Lower end of tibia. Illustrated in *Atlas* No. 39 p. 121.
- 33 *St. Bartholomew's Hospital* A661 — Lower end of tibia. The tumour contains multiple cysts and microscopic examination shows the structure of an osteoclastoma. (No history.)
- 34 *Hunterian Museum* R.C.S. 7401 — Lower end of femur. Illustrated in *Atlas* No. 39 p. 123.
- 35 *Hunterian Museum* R.C.S. 19711 — Upper end of tibia. Illustrated in *Atlas* No. 38 p. 105.
- 36 *Hunterian Museum* R.C.S. 19721 — Lower end of tibia. Illustrated in *Atlas* No. 38 p. 103.
- 37 *Guy's Hospital* 4422 — Shaft of femur. Illustrated in *Atlas* No. 38 p. 101.
- 38 *St. Bartholomew's Hospital* A680 — Intravascular osteoclastoma of tibia. The tumour consisting of an encapsulated reddish brown tissue had been found adherent to the superficial aspect of the periosteum of the middle of the subcutaneous surface of the tibia, whose impression is upon one side of the specimen. No bone or cartilage is present. Microscopically the structure is that of an osteoclastoma containing collections of pigment granules lying here and there in the fibrous stroma. The capsule is well defined and fibrous. Man, aged 37. Six months previously a swelling $\frac{1}{2}$ in diameter had been noticed on the skin—it gradually enlarged but remained painless.

FOCAL FIBROSIS OF BONE

- 39 *Guy's Hospital* 4433A¹ — Rib. A portion of a rib split showing a spindle shaped enlargement, 15 cm. long and 5 cm. in width, occupied by a dense mottled grey material which was found on microscopical examination to consist of fibrous tissue containing spicules of bone which is unusually cellular though not having a conspicuous margin of osteoblasts. Man, aged 31.
- 40 *London Hospital*, 160/1927 — Rib. A portion, 5.5 cm. long, of the 11th rib in the posterior axillary line, showing a fusiform expansion 2.5 cm. \times 1.5 cm. occupied by a dense grey tissue containing central pigmentation from hemorrhage. Microscopical examination shows the centre of the enlargement to consist of fibroblastic tissue containing hemorrhages. Peripherally this merges with vascular fibrous tissue.

FIBROCYSTIC DISEASE OF BONE—CATALOGUE

- containing spicules of bone and osteoid tissue. The 'expanded' cortex consists of new bone and osteoid tissue with irregular cartilage in places. Appositional osteogenesis is prominent, and osteoclasts are scanty. Boy, aged 19. Eighteen months injury followed by swelling which subsided, twelve months, similar injury, six weeks, painless swelling.
- 41 *Guy's Hospital*, 4133A²—Upper end of fibula, showing expansion by a central mass, which was found microscopically to consist of dense fibrous tissue containing numerous small spicules of bone. The affected area is separated from the upper epiphysis by a zone of irregular ossification. Girl, aged 12.
- 42 *Cancer Hospital*, 1578—Mandible. The greater part of the ramus and body of the right mandible expanded by a grey material, which proved on microscopic examination to consist of dense fibrous tissue containing in places bone lamellae which are not outlined by osteoblasts. Man, aged 30.
- 43 *St George's Hospital*, 239AV—Mandible. One half of the body of the mandible the normal tissue of which is replaced by a dense, homogeneous tissue containing a softened zone, $\frac{1}{2}$ in in diameter, at the apex of one tooth. Microscopic examination shows an abscess in this situation, with a few strips of epithelium. The remainder of the mass consists of fibrous tissue containing spicules of bone and osteoid tissue, osteogenesis is apparent in places, osteoclasts are not seen. (The changes appear to be too extensive to represent a response to the presence of the abscess.) Boy, aged 19.
- 44 *Cancer Hospital*, 1181—Spine. Softened vertebra from the mid dorsal region. Microscopic examination shows fibrous tissue containing scattered bone lamellae some of them outlined by osteoblasts. Boy, aged 19. Ten months' gradual onset of paraplegia, temporarily ameliorated by laminectomy.
- 45 *Guy's Hospital*, 4419—Tibia. Illustrated in *Atlas* No 38, p 409.
- 46 *St Bartholomew's Hospital*, A395—Shaft of tibia. A portion, 11 cm long, of the shaft of the tibia rather above its centre. The bone is occupied by a solid, dense mottled tissue over the surface of which the thinned cortex may be traced. Cysts are absent. Microscopic examination shows fibrous tissue containing numerous spicules of bone, most of these appear inactive, but osteoblastic activity is present in places, and lacunar absorption in others. In parts the fibrous tissue shows a myxomatous change. Boy, aged 13. Painless swelling noticed for two weeks.
- 47 *Guy's Hospital*, 4133B₁—Scapula. The angle and adjacent portion of a scapula, the posterior aspect of which is occupied by a mass of dense tissue, honeycombed with cysts varying in diameter up to 3 cm. Microscopic examination shows a spindle cell tissue, containing in parts extremely numerous small empty cysts giving it a sponge like appearance, and elsewhere networks of newly formed bone and masses of highly cellular cartilage. Man, aged 27. Symptomless swelling. (Note: Sirecoma is a possible alternative interpretation of this specimen.)
- 48 *King's College Hospital*, A774—Focal fibrosis, followed by spindle cell sarcoma. The upper half of a femur, split. In the subtrochanteric region its continuity is interrupted by a growth which is both endosteal and subperiosteal. The cut surface is smooth, compact, and white. Microscopic examination shows a spindle cell sarcoma containing giant cells of malignant type. Man, aged 45. Eighteen months previously a zone of diminished radiographic density at this site had been excised, the material removed being said to consist of a fairly dense fibrous tissue containing very many small irregular areas of ossification. (The patient is alive and well twenty nine months after disarticulation at the hip joint.)

DIFFUSE FIBROSIS OF BONE

- 49 *Hunterian Museum, R C S*, 7408—Tibia and fibula. Illustrated in *Atlas* No 39, p 431.
- 50 *Hunterian Museum, R C S*, 7405—Tibia and fibula. Illustrated in *Atlas* No 38, p 411.

FIBROCYSTIC DISEASE OF BONE—CATALOGUE

- 51 *University College Hospital* 79A3 and 79A4—Tibia and fibula from a child. The tibia is curved and expanded in places, the expansions being occupied by masses of solid tissue with a definite outline replacing the medulla and partly destroying the cortex. The fibula shows a similar expansion. The tissue consists of irregular trabeculae of bone separated by spaces containing cellular fibrous tissue. Boy, aged 3 (Bowing of the leg had begun at the age of 5 months.)
- 52 *Hunterian Museum R.C.S.* 3810 1—Tibia. The unacrated skeleton of the foot and lower half of the leg. The tibia is uniformly bowed forwards, the fibula shows a pseudo arthrosis in the lower quarter, the two fragments themselves straight meeting here at a right angle. Man, aged 23. (First symptom, prominence of the lower part of the tibia at 18 months.)
- 53 *Hunterian Museum R.C.S.* 740 1—Tibia. The inner half of the right tibia of an adult. The shaft is thickened and bowed. Microscopical examination shows that the cancellous bone is extensively replaced by fibrous tissue containing islands of bone substance which show in some places osteoclastic absorption and in others osteogenesis, many are outlined by osteoblasts. At the margin the cancellous bone shows little change except for fine fibrosis of the marrow, where it abuts upon the zone of gross change extensive osteoclastic activity is apparent. The fibula is unaffected except by slight bowing. Woman, aged 65. (Bowing and thickening dated from an injury in early childhood.)

HYPERPARATHYROID TYPE OF FIBROCYSTIC DISEASE OF BONE

- 54 *St Bartholomew's Hospital* A129 and A130—Femora and humerus. The upper extremities of the femora and of one humerus of a case described as osteomyelitis with myeloid tumours. There is a fracture of the neck of the left femur and cystic cavities in all three bones. Sections of the wall of one of the cysts show a structure resembling an osteoclastoma. The long bones were softened, and the skull so soft that it could be bent with ease. Osteoclastomata were present in one rib and in the lower jaw. Patient, aged 50 (1881).
- 55 *Gay's Hospital*, 443C—Femur and tibia showing bending and fracture. In the femur there are cystic cavities and osteoclastomata. Microscopical examination shows zones of osteoclastomatous tissue intersected by fibrous bands containing bone lamellae outlined by osteoblasts. Woman, aged 60 with multiple pathological fractures.
- 56 *Charing Cross Hospital* 1141—Femur, tibia, and fibula thickened and bent with much softening and the formation of cystic spaces. (No history.)
- 57 *Hunterian Museum, R.C.S.*, 3808 1, 3808 2, 3808 3, 3808 4, and 3808 5—Culcrum, femora, and tibia. Illustrated (3808 1, 3808 3, and 3808 4) in *Atlas* No. 39, pp. 433, 435, and 437.
- 58 *Middlesex Hospital*, C79 and C79A—Femur and humerus. Right femur showing severe deformity, and a distension of the shaft by a hemorrhagic cystic tumour, upper end of the left humerus. Woman, aged 43, with spontaneous fractures of the left femur and left humerus, and general bone deformities. Onset with pathological fracture of the left femur, two years before death (1921).
- 59 *London Hospital*, 233/1929 and 231/1929—Bones and parathyroid tumour. Femur showing spontaneous fracture through a large cyst. There are other cysts at the lower end and an osteoclastomatous nodule, the cortical bone is thinned. Humerus, sternum, and ribs from the same case, showing porosity of the cortical layer and nodular expansion of the bone. Man, aged 66. Two years' history of pain in bones and joints, pathological fractures of the left femur and the proximal phalanx of the right middle finger.
- 60 *London Hospital*, 60/1929—Normal parathyroid glands. Dissection to show the situation of normal parathyroid glands.
- 61 *London Hospital*, 489/1930 and 482/1930—Parathyroid tumours, renal calculi, humerus, and skull. Illustrated in *Atlas* No. 48, pp. 413, 415, and 416.

FIBROCYSTIC DISEASE OF BONE—CATALOGUE

- 62 *London Hospital* 506/1929 —Parathyroid tumour Woman, aged 41
- 63 *London Hospital* 317/1930 —Parathyroid tumour Woman aged 37
- 64 *London Hospital* 379/1930 —Parathyroid tumour Woman aged 49
- 65 *London Hospital*, 531/1930 —Parathyroid tumour and adenoma of thyroid Woman, aged 51
- 66 *London Hospital*, 369/1931 —Parathyroid tumour Woman aged 25
- 67 *London Hospital* 66/1932 —Parathyroid tumour Woman aged 69
- 68 *London Hospital*, 12/1933 —Parathyroid tumour Woman aged 20
- 69 *London Hospital* 65/1933 —Parathyroid tumour from the mediastinum Woman aged 22
- 70 *Hunterian Museum R C S* 740.9 and 740.91 *Middlesex Hospital* C 79.1 and C 79.1A —Bones and parathyroid tumour from a case of hyperparathyroidism Girl, aged 20 (G. Gordon Taylor, Philip Wiles, and S. L. Baker *British Journal of Surgery* 1932 viii, 606.)
- 71 *Cancer Hospital* 1562 —Parathyroid tumour from a woman aged 40 who had an osteoclastoma of the jaw, and slight general softening of the bones
- 72 *Cancer Hospital* 1576 —
 - a Rarified portion of tibia. Microscopical examination shows zones of osteoclastomata tissue intersected by fibrous tissue containing bone lamellae outlined by osteoblasts and showing lacunar absorption in a few parts
 - b Parathyroid tumour from the same case Woman aged 58 (A. L. Abel, G. Thomson, and I. M. Hawksley *Lancet*, 1933, ii 525)

SARCOMA OF MUSCLE



Section through part of a vastus externus muscle

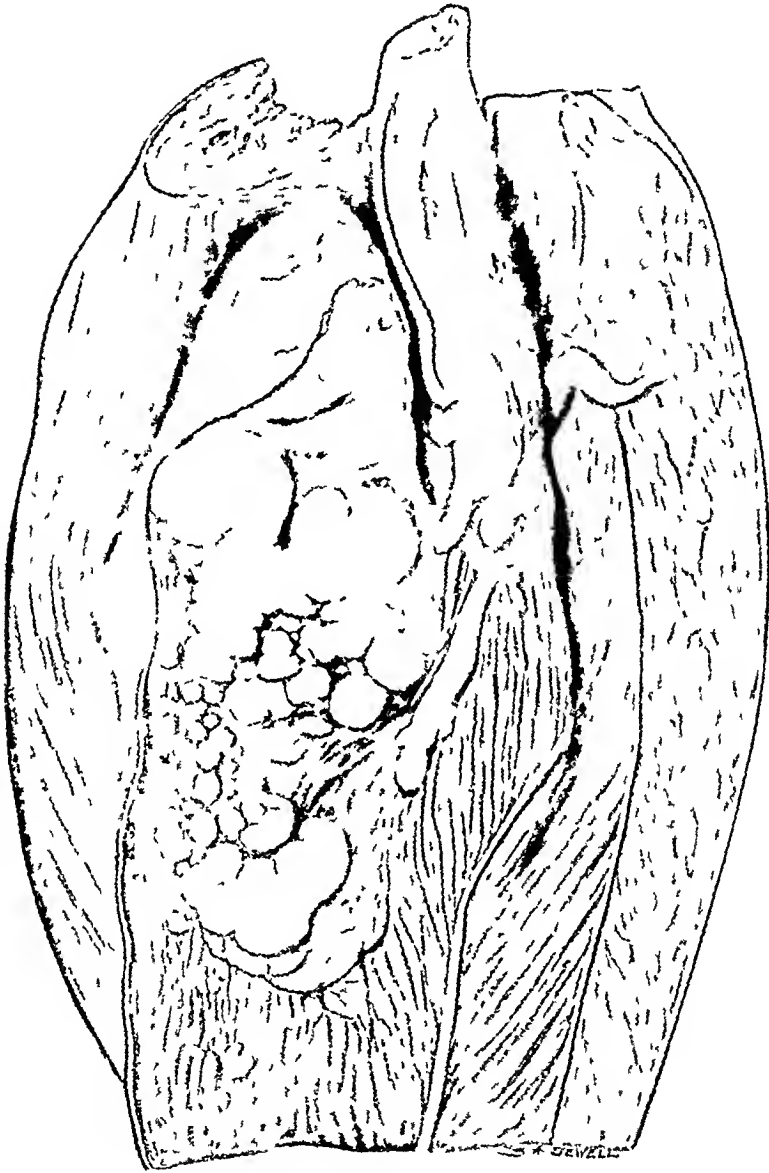
The muscle contains a tumour about 2 in in diameter, pale yellow in colour, and containing numerous small cysts which in the fresh state, were filled with brown serous fluid. Above and to the left the growth is surrounded by an imperfect capsule, below and to the right it is infiltrating the muscle.

Museum of the College of Medicine, University of Durham, Newcastle upon Tyne, 633/6

MICROSCOPIC STRUCTURE—Spindle-celled sarcoma

CLINICAL HISTORY—The patient was a girl, aged 13 years, who had noticed a lump on the outer side of the right thigh three weeks before admission to hospital. On examination the swelling was fluctuant but was not attached to the skin. It was removed by operation, together with the layer of healthy muscle surrounding it. It recurred locally two years later and was again removed.

SARCOMA OF MUSCLE INFILTRATING VEINS



Part of a triceps suræ

From the back of the specimen a V-shaped piece has been removed to show a sarcoma originating in the soleus. On the front the commencement of the popliteal vein has been laid open by a coronal section. The tumour has invaded the tibial and popliteal veins, the lumen of which is entirely occupied by the white opaque growth.

Museum of University College Hospital, 104

MICROSCOPIC STRUCTURE—Round-celled sarcoma

CLINICAL HISTORY—See p 447

SARCOMA OF MUSCLE EXTENDING ALONG A VEIN

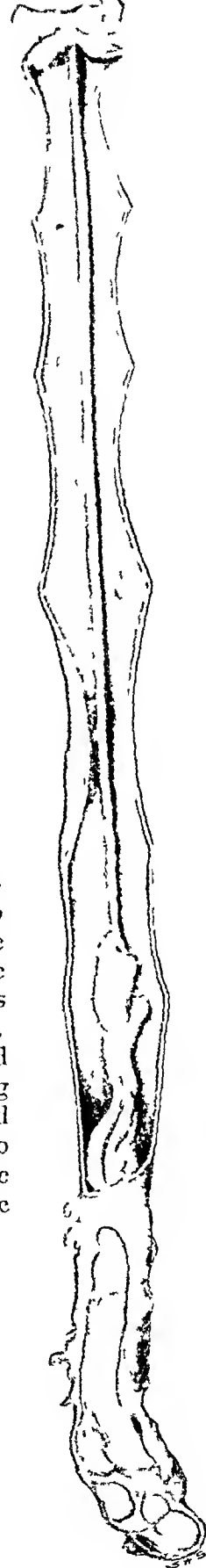
The popliteal and femoral veins from the previous case

The popliteal vein is completely plugged by a solid white growth a continuation of which in the form of a narrow ribbon lies loose in the lumen of the femoral vein. There is a small thrombus round the lower end of the narrow ribbon of growth.

Museum of University College Hospital 105

MICROSCOPIC STRUCTURE —Round-celled sarcoma

CLINICAL HISTORY —The patient was a girl aged 17 who had had a painful swelling of the left calf for two months. There was a movable elastic swelling in the muscles of the left calf with distended veins in the overlying skin, which was hot but not red or oedematous. No tenderness or pulsation in the tumour. The glands in the groin were not enlarged, nor was there any interference with the movements of the ankle-joint. An incision was made into the calf and clear fluid came out from a cavity in the growth. Amputation above knee. The lower end of the femoral vein contained blood-clot, and on pulling this out a long, thin white thread was found attached to it. When laid along the thigh this thread reached to just above Poupart's ligament. It was thought to be growth, and disarticulation was performed at the hip. The patient died shortly after operation.



HYDATID CYST OF LIVER

The right lobe of a liver in sagittal section

An hydatid cyst projects upwards from the superior surface of the liver from which it is separated by its fibrous capsule. The outside of the cyst is roughened by the fibrous adhesions which united it to the diaphragm. Its lining is granular. Inside the cavity are innumerable daughter cysts with thin translucent walls. They contain a watery fluid in which is a copious granular deposit of a deep orange colour. The cut surface of the liver is pale and fibrotic.

Museum of the Manchester Royal Infirmary 221

CLINICAL HISTORY—The patient was a woman aged 44 who during the three months before her death was treated for cirrhosis of the liver with ascites and œdema of the back and legs. Paracentesis abdominis was performed repeatedly. The liver was hard and reached almost to the level of the umbilicus.

Blood-count—

Red cells	3 104 000	Hb 62 per cent
White cells	1 000	
Polymorphs		59.5 per cent
Large mononuclears		25.5
Small mononuclears		10.5
Eosinophils		1.5
Transitional cells		3.5

Wassermann reaction—Negative

AUTOPSY—The diaphragm was at the level of the 2nd intercostal space in the mid-clavicular line, and was firmly adherent to the compressed lower lobe of the right lung. The cirrhotic liver was adherent to its lower surface and to the adjacent viscera. In the upper and posterior part of the liver were three hydatid cysts. The peritoneal cavity contained sero-purulent fluid which had been infected through perforation of an ulcer in the skin covering an umbilical hernia. Microscopic examination of the liver showed an irregular interlobular and intercellular fibrosis.

HYDATID CYST OF LIVER



MUSEUM OF THE MANCHESTER ROYAL INFIRMARY, 221

NO 40—SUPPLEMENT

U 1

GUMMA OF SPLEEN

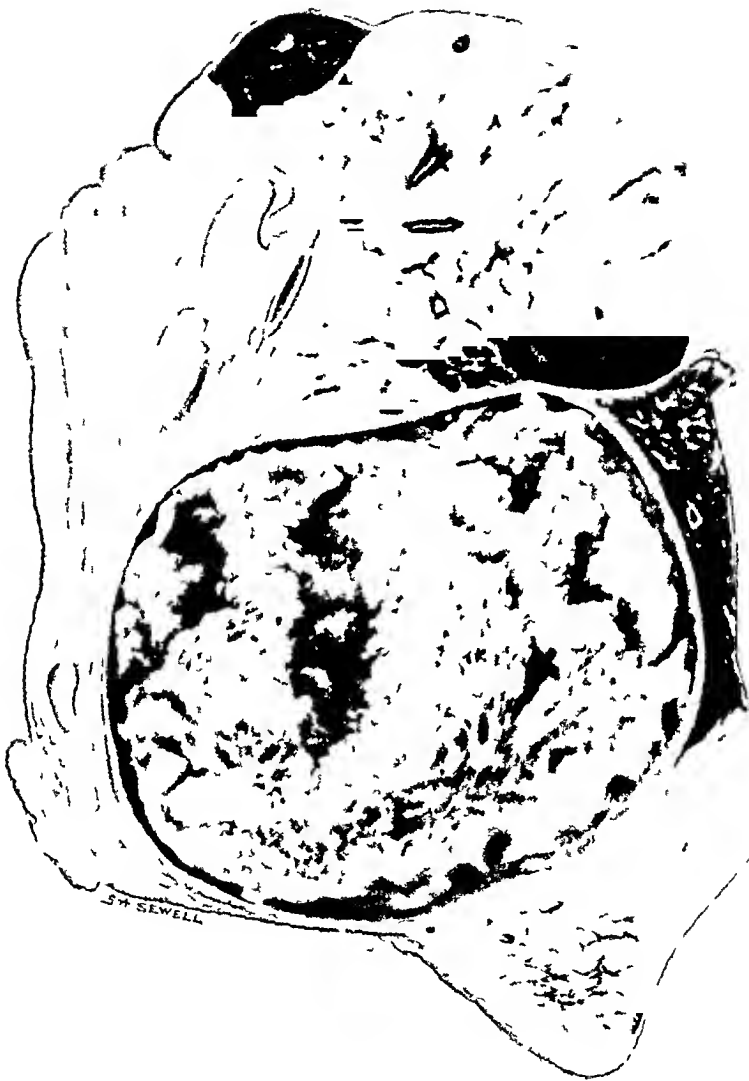
The cut surface of a divided spleen

The spleen is enlarged, the capsule is thickened, the parenchyma is slightly paler than usual and there is some overgrowth of the fibrous reticulum. A rounded gumma, about $2\frac{1}{2}$ in in diameter occupies the lower half of the organ. The gumma is surrounded by a dense fibrous capsule and is of the colour and consistency of the yolk of a hard-boiled egg.

*Museum of the College of Medicine, University of Durham
Newcastle upon Tyne, 565/10*

CLINICAL HISTORY.—The patient was a woman aged 44 who died as the result of an accident.

GUMMA OF SPLEEN



MUSEUM OF THE COLLEGE OF MEDICINE UNIVERSITY OF DURHAM, NEWCASTLE UPON TYNE, 565/10

GUMMA OF LIVER

Part of a liver divided by longitudinal section

A large, yellow, structureless area extends from the hilus into the liver

Pathological Museum, Victoria University Manchester 2453

CRITICAL HISTORY —The patient was a single woman aged 20 who was admitted to hospital in a moribund condition with a history of persistent vomiting for ten weeks. She complained of pain in the right hypochondrium. She had never menstruated since the age of 14.

On examination she was too ill to give a history and was emaciated with a dry and scaly skin, hollow cheeks and sunken eyes. The tension in the eyeballs was low. The urine contained a trace of albumin and diacetic acid. There were no physical signs of disease in chest or abdomen. There was no jaundice nor any pigmentation of the skin.

She died two days after admission.

AUTOPSY —Fibrous stricture of pylorus which admitted a probe with difficulty. Liver enlarged and surface mottled by large pale areas.

On section there was a gumma in the right lobe $2\frac{1}{2}$ in in long diameter. Its margins were sharply limited by a layer of connective tissue. The surrounding liver showed chronic congestion and fatty infiltration. Uterus and ovaries very small.

Atheroma of ascending aorta.

GUMMA OF LIVER



PATHOLOGICAL MUSEUM, VICTORIA UNIVERSITY, MANCHESTER, 24 83

SEBACEOUS ADENOMA OF SCALP

A section through a tumour with the overlying portion of scalp

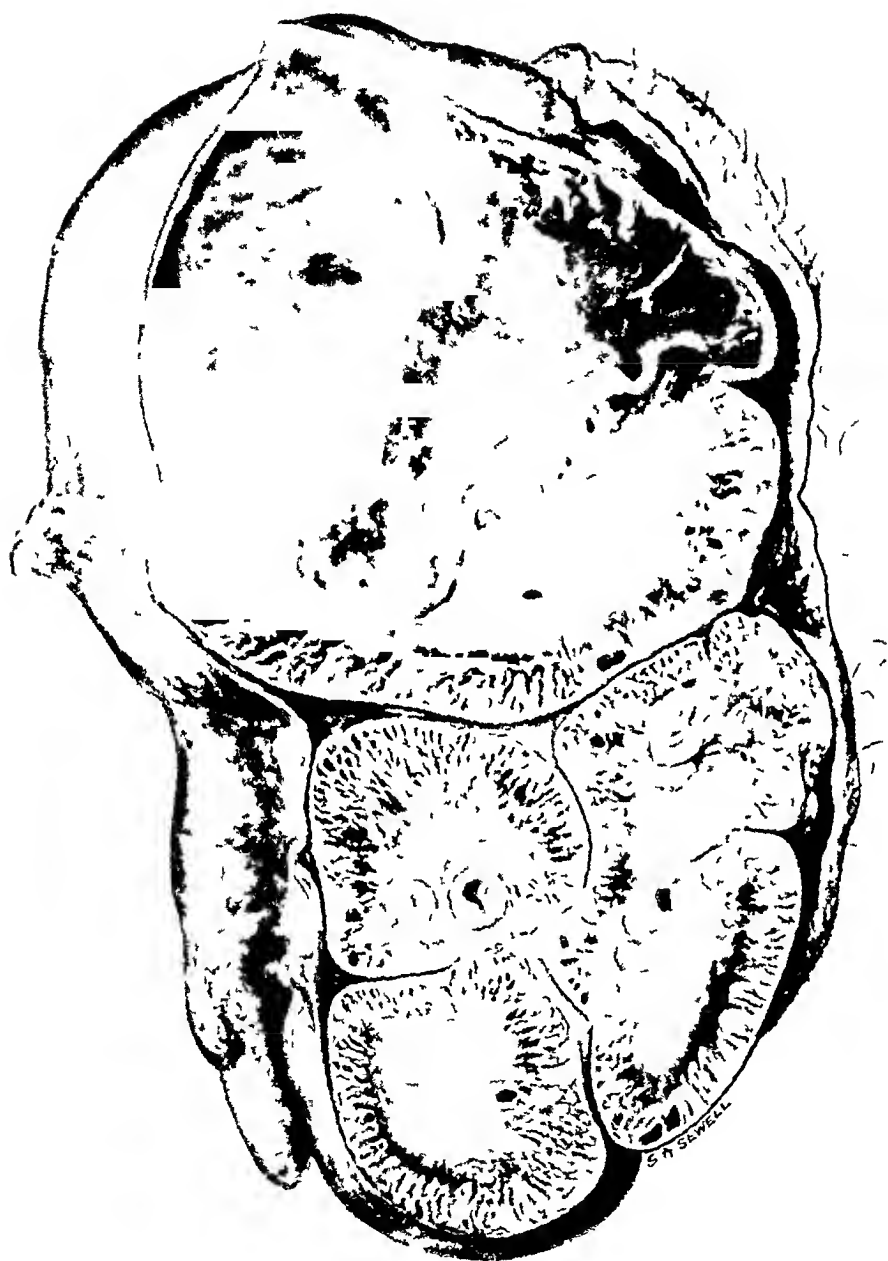
The tumour measures 5 m. in its long diameter. Its upper part is cystic and was filled in the recent state with modified sebaceous material. The remainder of the tumour is solid and consists of connective tissue studded here and there with epithelial remnants. The scalp overlying the tumour is atrophic, and has an ulcer 2½ m. in diameter.

*Museum of the College of Medicine, University of Durham
Newcastle upon Tyne, 503/5*

MICROSCOPIC STRUCTURE.—The tumour is encapsuled and is composed of papillomata with a thick coating of keratin.

CLINICAL HISTORY.—The patient was a woman aged 55 who had had a sebaceous cyst of the scalp as long as she could remember. For some time before its removal by operation it had caused much inconvenience by reason of its size and of the ulcer on its surface. Many members of her family were troubled with sebaceous cysts.

SEBACEOUS ADENOMA OF SCALP



MUSEUM OF THE COLLEGE OF MEDICINE, UNIVERSITY OF DURHAM NEWCASTLE UPON TYNE, 502/5

INTESTINAL OBSTRUCTION BY GALL-STONE



A coil of small intestine

Part of the intestine has been opened to show a large oval gall-stone which is impacted in the lumen. The bowel above the obstruction is distended and engorged.

Pathological Museum, University of Birmingham, 7386

No clinical history

STRANGULATED OBTURATOR HERNIA

The pubic and ischial portions of an innominate bone with the soft parts covering its internal surface (p. 158)

From the obturator foramen projects a hernial sac congested at its tip. Behind and to its outer side run the obturator vessels with the posterior division of the obturator nerve. The anterior division of the obturator nerve passes to the outer side and in front of the sac. On the peritoneal aspect the entrance to the sac is narrowed by congestion of the surrounding soft parts.

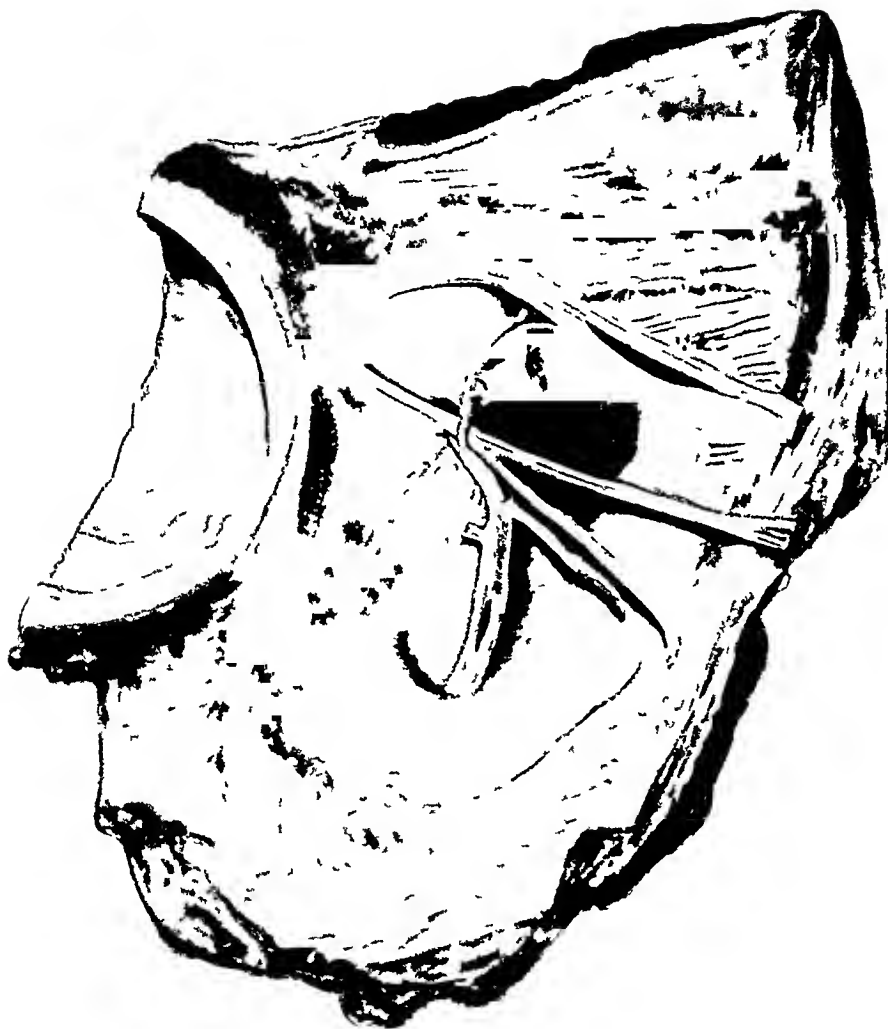
Museum of University College Hospital, 23A4

CLINICAL HISTORY—The patient was a woman aged 71 who was admitted to hospital with a history of eight days' intestinal obstruction without pain. The abdomen was distended by visible coils of small intestine and there was frequent stercoraceous vomiting. Laparotomy revealed strangulation of the apex of a loop of small intestine about one foot above the ileocecal valve by a right obturator hernia. The hernia was reduced without difficulty, the coil of intestine being congested but viable. There was no corresponding sac on the left side.

Death nine hours after operation.

AUTOPSY—No peritonitis. The small intestine was empty below the site of strangulation, distended up to the pylorus above.

STRANGULATED OBTURATOR HERNIA



SHOWING OUTSIDE OF SAC

MUSEUM OF UNIVERSITY COLLEGE HOSPITAL, 23Y4

STRANGULATED OBTURATOR HERNIA.



The loop of small intestine which was strangulated in the sac of the preceding specimen, 23Y4

The strangulated portion is marked off by a groove, best defined on the proximal side. Within the area bounded by this groove the wall of the bowel still shows some trace of the intense congestion which was present at the time of operation. The gut on the proximal side of the obstruction is dilated and congested, that on the distal side is normal.

Museum of University College Hospital, 23Y5

STRANGULATED VENTRAL HERNIA

The sac of a ventral hernia with its contents

Two portions of the bowel enter the hernia one being small intestine and the other transverse colon. The outer part of the sac is filled by omentum and congested bowel occupies the centre.

Museum of St. Bartholomew's Hospital, M 196

CLINICAL HISTORY —The patient was a woman, aged 53, who had had a ventral hernia below the umbilicus for several years. For one year it had been irreducible, and for one week there had been symptoms of strangulation. She died under the anæsthetic before the operation was begun.

STRANGULATED VENTRAL HERNIA



VOLVULUS OF SMALL INTESTINE

A coil of small intestine

The twisted bowel is congested to a deep purple colour and is moderately distended

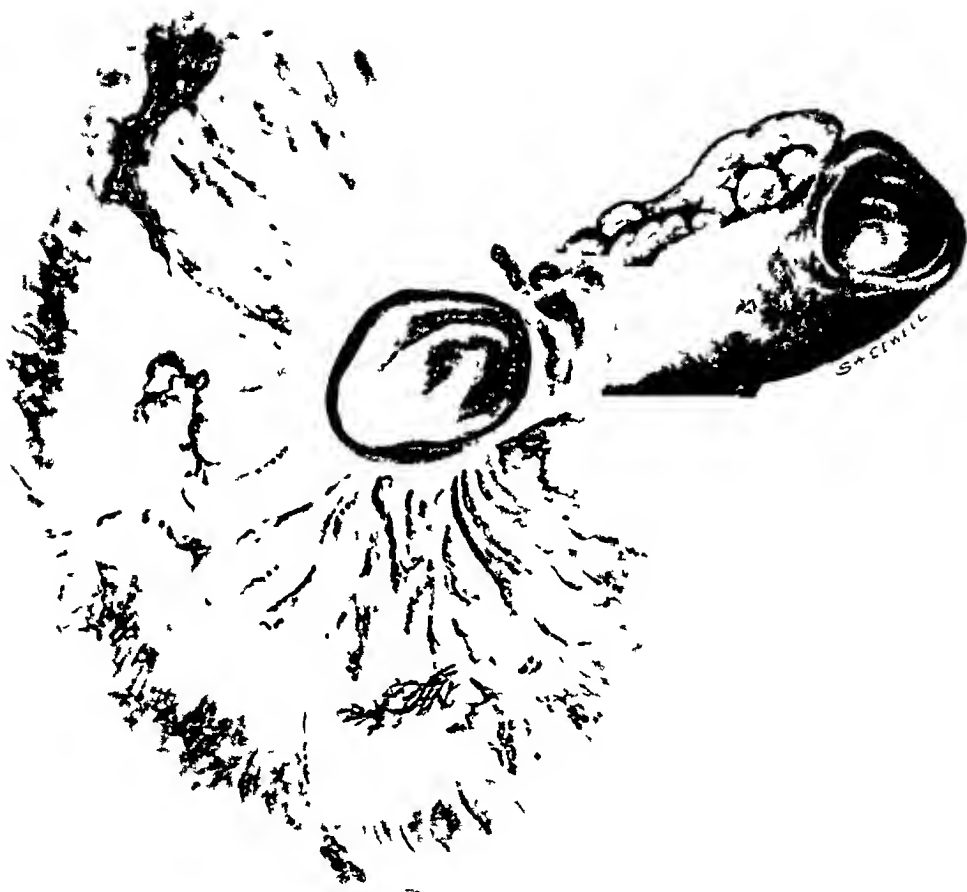
Hunterian Museum, R C S, 6351

CLINICAL HISTORY —The patient was a woman aged 42. Four years before her fatal illness she had had her left kidney removed on account of suppuration, and shortly afterwards four gall-stones were taken out of the gall-bladder.

The onset of the volvulus was marked by severe abdominal pain and vomiting. A very tender swelling was felt on the right side of the abdomen on the following day. The abdomen was opened on the third day and the specimen shown was removed, the cut ends of the intestine being stitched into the wound. There were some old adhesions between the bowel near the twist and the region of the gall-bladder.

The patient continued to vomit from time to time and died eight days after the operation. Faeces never passed freely from either of the fistulae.

VOLVULUS OF SMALL INTESTINE



HUNTERIAN MUSEUM RCS 6315 1

ILEO-CÆCAL INTUSSUSCEPTION

The terminal part of the ileum the cæcum and the proximal half of the ascending colon, divided by coronal section

The intussusepted portion begins at the ileo-cæcal valve and the appendix, which has been drawn in with it has been deflected from its natural position between the middle and inner layers of the intussusception to make its relations clear. The intussusceptum is very œdematous and the œdema extends to the submucosa of part of the bowel outside the strangulated area. The intussuseipiens is not so much affected but some congestion is present and a few flakes of lymph are seen on the surface of the appendix.

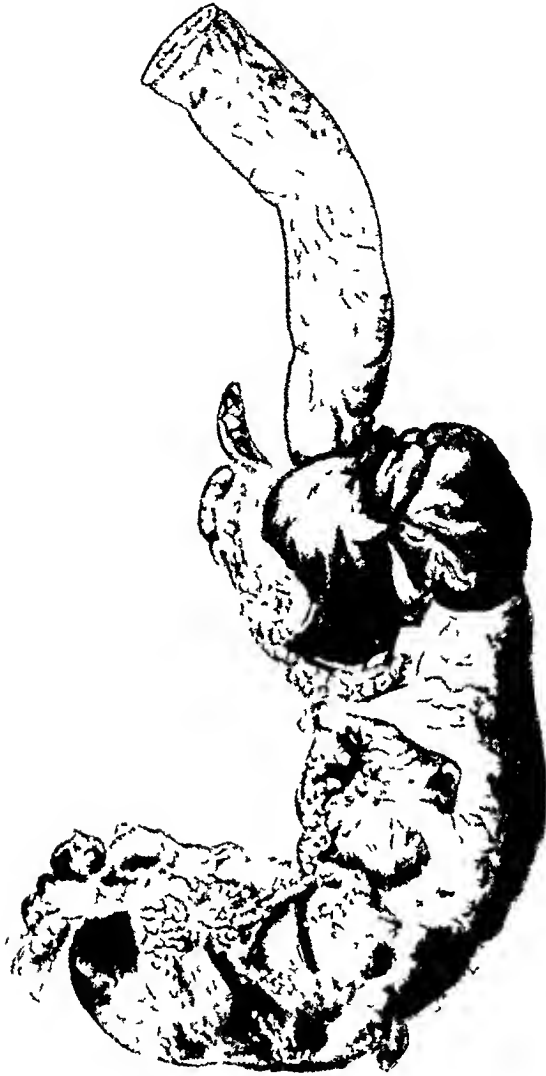
Museum of St Bartholomew's Hospital, M 215

CLINICAL HISTORY.—The patient was a man aged 35 who came to hospital with acute intestinal obstruction. He died a few days later of peritonitis and broncho-pneumonia.

ILEO CÆCAL INTUSSUSCEPTION



ILEO CAECAL INTUSSUSCEPTION



Portions of the small and large bowel in which an intussusception has developed

The intussusception is of the ileo-caecal variety. Part of the appendix has disappeared into the intussusception the distal portion alone being visible by the side of the entering coil. Near the neck of the intussusception the receiving layer has been ruptured during an attempt at reduction and the intussuscepted portion protrudes through the hole.

Museum of the University of Sheffield, I XXXIII-13

CLINICAL HISTORY—The patient was a woman who had been passing blood by the rectum for thirty hours before admission to hospital. She vomited eight hours after the onset of symptoms. At operation the specimen shown was resected after an attempt to reduce the intussusception had caused rupture of the outer coat. The wound in the abdominal wall gave way eight days after operation and was re-sutured. The patient recovered.

MECKEL'S DIVERTICULUM FROM A STRANGULATED FEMORAL HERNIA

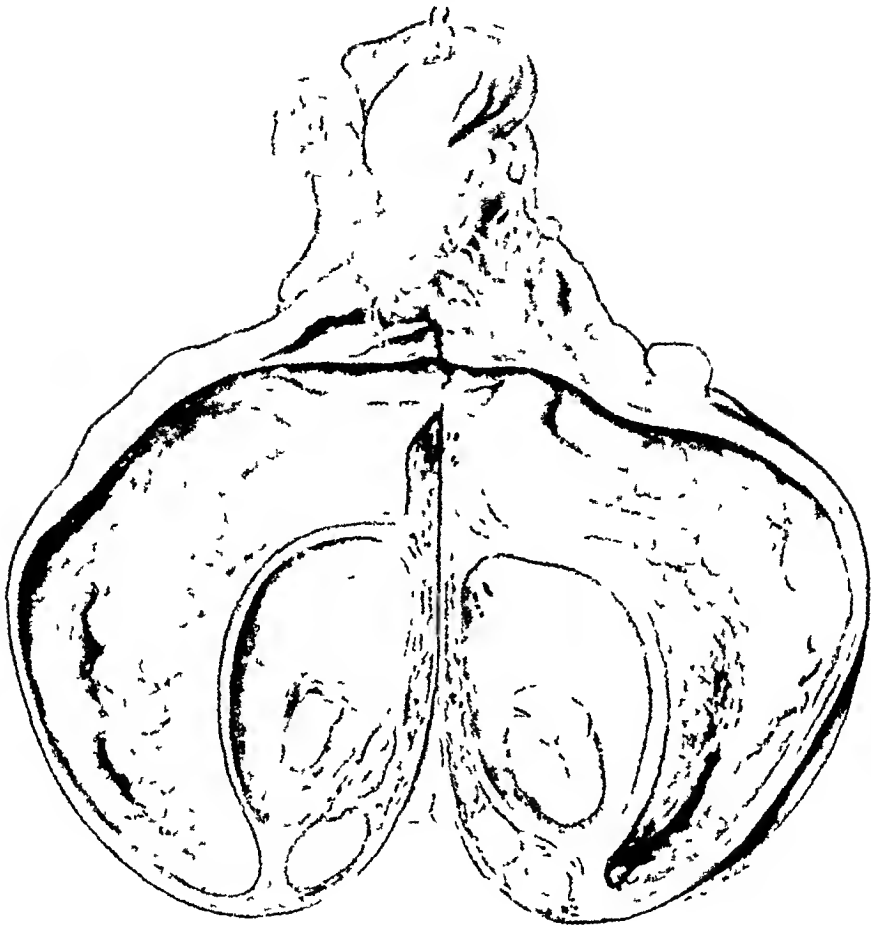


A Meckel's diverticulum $1\frac{1}{2}$ in long, with the adjoining part of the small intestine, which had become strangulated in a femoral hernia. The whole of the diverticulum with about 3 in of the adjoining ileum is of a dark purple colour.

Hunterian Museum, R C S 6781 1

CLINICAL HISTORY—The patient was a woman, aged 50, who had had a femoral hernia for two years. It had been strangulated for six hours before admission to hospital.

At operation, the sac was found to contain faecal-smelling fluid, and on removal of this a black object, about the size of the little finger, came into view. This was found to be connected with the bowel, a portion of which was also blackened. The gangrenous portion of bowel containing the Meckel's diverticulum was excised, and a lateral anastomosis made. The bowel was healthy and was not distended either above or below the injured part.



The specimen consists of a testicle removed at necropsy

In the lower part of the testicle is an abscess, and in the lower pole of the epididymis a nodule of purulent inflammation

The tunica vaginalis is distended, and lined by opaque, greenish grey pus and fibrin

Museum of University College Hospital, 7BIII

MICROSCOPIC STRUCTURE — Purulent inflammation of tunica vaginalis
Abscess in testicle

CLINICAL HISTORY — The patient was a man, aged 52 who was admitted to hospital with a perineal abscess. He had suffered for many years from urethral stricture which followed gonorrhoea contracted at the age of 27. During the five years preceding admission external urethrotomy with removal of prostatic calculi, internal urethrotomy, and prostatotomy had been performed on different occasions.

The perineal abscess was opened, but later there was retention of urine needing daily catheterization. An instrument of size 6 F was passed and drew off urine containing thick pus. An acute tender swelling of the right testicle developed, the temperature rose to 101° F, and there were rigors. External urethrotomy was again performed and an abscess was found in the bulb. The patient died of urinary infection and renal insufficiency thirty-five days after admission to hospital.

